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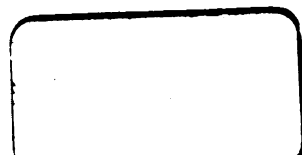
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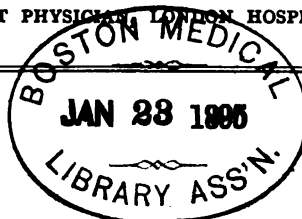
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THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 11, 1893.

TWO CLINICAL LECTURES ON TYPES OF PULMONARY PHTHISIS IN ADULTS.

Delivered in connection with the London Post-Graduate Course at the Brompton Hospital for Consumption and Diseases of the Chest,

By **PEROY KIDD, M.D., F.R.O.P.,**

Physician to the Hospital; Assistant-Physician and Pathologist to the London Hospital.

We are all only too familiar with the common form of phthisis, or pulmonary tuberculosis, an affection which may briefly be defined as a *progressive consolidation of the lung, commencing in the upper lobes, and tending to undergo ulcerative softening and excavation, associated with wasting of the body, and other symptoms of failure of the general health.*

But, in certain cases, pulmonary tuberculosis presents features which render its recognition a matter of some difficulty. I propose, therefore, to speak briefly about the principal clinical types which the disease assumes, illustrating my remarks by a demonstration of cases which I shall ask you to examine for yourselves.

Before entering on the topic of classification, I must say a few words concerning the

MORBID ANATOMY OF PULMONARY TUBERCULOSIS;

for without clear ideas on this subject, it is impossible to grasp the meaning of the complex physical signs at times presented by patients suffering from this disease.

The primitive typical lesion in its earlier stage takes the form of a small nodule, or, less commonly, of several discrete nodules situated in the apex of one or both lungs. In either case crops of similar nodules gradually spring up in the neighbourhood of the primary lesion, and thus racemose, or grape-shaped areas of consolidation are developed. Where the disease is chronic these isolated foci tend to coalesce, so that a considerable section of the lung is invaded and rendered airless. In rare cases a large number of lobules, or even a whole

lobe, may be simultaneously involved without a previous nodular stage, though a combination of the two lesions is less uncommon.

I shall not attempt to enter into the minute anatomy of tubercular lesions, merely stating that the nodules or larger tracts of consolidation consist of a peculiar cellular growth, dependent on the presence of a special microbe, the tubercle bacillus.

If we examine the earliest tubercular lesion, we discover that it consists essentially of an islet of broncho-pneumonia, that is to say, the process starts from a small bronchus and its corresponding lobule or lobules. The tubercular growth is peculiar, not only in structure, but in its pathological destiny. The central or oldest part of the nodule undergoes a slow necrosis, involving all the cellular elements which lose their outline and gradually become fused into an amorphous, dry, cheesy, or caseous mass. Outside this zone, at the margin of the necrotic area, is a zone of proliferating tubercular cells, in and among which the tubercle bacilli are in active growth. At the external margin of the tubercular lesion, organization of cells into connective tissue is taking place, whereby a species of capsule is developed. Where healing ensues this external connective tissue zone acts as a barrier, preventing the extension of the disease, the central caseous part being enclosed in a definite capsule. The caseous material at other times becomes infiltrated with lime salts, or is converted into a knot of fibrous tissue, and thus the morbid process is brought to an end.

In the more common case where extension of the disease occurs, the development of the external connective tissue zone does not keep pace with the growth of tubercle cells within, which, consequently, spread the disease step by step into the tissues around.

In man the processes of necrosis, cell-growth, and organization are commonly associated in varying proportions. According as the fibrous or caseous element predominates, the tendency is towards arrest or destruction. In the latter case, softening of the necrotic mass takes place, followed by ulceration into a bronchus, and a cavity is then formed. But it is important to remember that while the disease is manifesting fibrous or reparative

changes at one centre, necrosis and excavation may be actively proceeding at another. As a result of the fibrous changes in the lung, various degrees of contraction occur leading to displacement of the heart towards the affected side, drawing up of the diaphragm with the abdominal viscera, and retraction of the chest wall. Another result of contraction of the lung is seen in a secondary compensatory enlargement of the alveolar cavities in the neighbourhood, and varying degrees of emphysema thus arise. In chronic cases where a large portion of one lung is infiltrated, a compensatory enlargement and eventually definite emphysematous changes are commonly developed in the opposite lung also.

A localized persistent catarrh of the smaller bronchi in the neighbourhood of the tubercular foci is an invariable concomitant of the process. The thickening of the bronchial mucous membrane and the increased secretion which result, give rise to obstruction of the affected tubes, in consequence of which the air in the affected lobule or lobules becomes absorbed, and lobular collapse ensues.

Pleurisy, whether circumscribed or diffuse, is an invariable consequence of progressive pulmonary tuberculosis. Fibrinous exudation is much more common than effusion of fluid. Direct extension of the pulmonary disease is the usual cause of pleurisy, though it may happen that pleural tuberculosis arises independently. Not unfrequently the pleurisy is unassociated with any recognizable tubercular lesion of the serous membrane. In some of these cases running a chronic course, the miliary tubercles that set up the pleurisy have probably undergone a fibrous change. At times the rapid extension of a softening process in the lung may involve the pleura, and lead to perforation and pneumothorax. But where the disease is chronic adhesions are formed sooner or later, and in old cases the amount of thickening of the pleura may reach a very extreme degree.

The bronchial glands are commonly involved in the morbid change, the tubercle bacilli being carried by the lymphatics to the corresponding glands, where they set up secondary tubercular processes. In children the glandular affection sometimes reaches such proportions that the bronchi become compressed and obstructed by the enlarged glands; but in adults this is hardly ever seen.

I show you here microscopical specimens illustrating (1) a small islet of tubercular bronchopneumonia; (2) tubercular bronchitis; (3) caseous

pneumonia; and, (4) a fibro-tubercular lesion of the lung, in which the development of connective tissue is especially well seen.

DISTRIBUTION OF LESIONS.

I must be content to state, without attempting to explain, the fact that the earliest lesion occurs at the apex of the upper lobe. Contrary to what might be expected the secondary lesions do not arise exclusively or mainly in the immediate vicinity of the primary focus, but generally at distant parts, more especially the apex and upper part of the lower lobe, and the lower and middle part of the upper lobe. The last parts to be involved are the base and anterior part of the lower lobe, and the extreme anterior inferior margin of the upper lobe.

METHODS OF EXTENSION IN THE LUNG.

The morbid process spreads in four ways:—

1. *By contiguity*, the tubercular growth extending step by step mainly along the preformed channels or lymph spaces.

2. *By the lymphatics*, the tubercle bacillus being carried by the lymph stream and deposited in some distant part of the lung or in the bronchial glands.

3. *By the blood vessels*. In certain cases the tubercular tissue projects into a branch of the pulmonary artery, in consequence of which the microbes escape into the circulation, and become impacted in the corresponding capillaries. But this is, comparatively speaking, an unimportant method of extension in the lungs.

4. Lastly, the disease is spread through the medium of *the bronchial tubes*. This is the most important of the four methods by which extension takes place. If you remember what I said about the distribution of the lesions, you will see that it is impossible to explain the early appearance of the secondary lesion in the apex of the lower lobe by a process of local extension; but if we recognize the part played by the bronchial tubes there is no difficulty in explaining this localization. It was shown by Dr. Ewart that the proclivity of this part of the lower lobe to the tubercular process depends on the fact that the apex is supplied by a large straight bronchus coming off horizontally from the main descending bronchus, and that infective particles are very liable to be carried through this large bronchus into the apex of the lower lobe. Where secondary lesions occur in this neighbourhood it will generally be found that there

is excavation at some part of the upper lobe discharging infective material into the respiratory passages. During the forcible inspiration which follows cough or exertion of any kind the secretion present in the bronchi is very liable to be sucked back into remote parts of the lung, and thus set up secondary foci of disease.

I turn now to the clinical side of the subject; and, in the first place, I wish to say a few words about the

MODE OF INVASION.

The Insidious Type.—In the greatest number of cases we shall find that the patient notices certain constitutional symptoms, such as wasting, debility, anæmia, dyspnoea on slight exertion, before characteristic pulmonary symptoms develop.

Bronchitic Type.—In another large group of cases the disease begins with a slight cough, which is often attributed by the patient to a common cold, constitutional symptoms developing subsequently, perhaps not for months. But in many cases which seem to begin with cough a little close questioning of the patient will show that a previous stage of debility or wasting had been passed through before the supervention of cough and expectoration.

Hæmoptoic Type.—In a much smaller number of cases, the “phthisis ab hæmoptoe” of the old authors, the first symptom to attract attention is hæmoptysis. Where the hæmoptysis is profuse it almost certainly indicates the existence of an aneurysm in a cavity, *i.e.*, of old-standing, though latent disease.

Pleuritic Type.—It is not very uncommon to find that the patient's illness began with a sudden pleuritic stitch. In such patients we may find, on examination, the signs of a local dry pleurisy, mostly in the axilla, without any further signs of disease of the lung. Nevertheless, it is probable that in these patients an apical lesion is also present.

Pneumonic Type.—This is the rarest mode of invasion. The patient is seized suddenly with a rigor, vomiting, pain in the side and dyspnoea, and he is at first believed to be suffering from acute croupous pneumonia; but, as a rule, the constitutional symptoms are less severe in the tubercular form, and the patient exhibits partial improvement in the course of a few days. Examination of the chest in such cases shows consolidation of a large part or the whole of one lobe at an early stage.

Pulmonary tuberculosis may be *acute* or *chronic*. Great difficulties are apt to arise in attempting to fix the date of onset, for, in many cases of chronic disease the patient is hardly conscious of being ill until, from some cause or other, the disease is started into activity. Moreover, many cases which begin acutely gradually lapse into a chronic stage. Nevertheless, the distinction of *acute* and *chronic* is not without value.

ACUTE FORMS.

1. *Broncho-pneumonic.*—In this variety the patient is attacked with symptoms of broncho-pneumonia, accompanied by marked constitutional symptoms, wasting, night-sweats, dyspnoea, and remittent pyrexia. The physical signs indicate a diffuse bronchitis, râles and rhonchi being scattered all over the chest. Later on, localized dulness or signs of pleurisy may arise in small patches, more especially in the upper lobes, and ultimately evidence of excavation may be discovered, though, as a rule, the patient succumbs before the signs of destruction of lung are sufficiently pronounced to enable us to detect them. On post-mortem examination we find softening tubercular masses and small cavities scattered through the lungs, separated by a varying amount of spongy tissue, which accounts for the slight evidence of consolidation obtainable during the patient's life. In cases running a most acute course it is not unusual to meet with chronic lesions of the apex, in the shape of an old capsulated cavity, acute dissemination having evidently resulted from infective cavity-secretion inhaled into distant bronchioles.

2. *Miliary Tuberculosis.*—This form is well-known with its marked dyspnoea, high temperature, cyanosis, and profound constitutional depression. Miliary tuberculosis may be part of a general tuberculosis, or, as we not uncommonly see in adults, it may start from a small old cavity which has given rise to no marked previous symptoms.

I can only briefly allude to the difficulty of diagnosing this affection from typhoid fever in its early stages, before the appearance of definite local symptoms.

3. *Pneumonic Form.*—I have already mentioned the pneumonic type of invasion; and I shall, therefore, merely say that while patients attacked with this form may be carried off in two or three months, at other times the disease quiets down, and runs a comparatively chronic course.

I will here briefly describe the case of a patient

who is at present in the hospital suffering from this form of the disease.

M. P——, a man, æt. 35, was admitted in August with the following history. He had always been well until he recently took to cloth portering. Two months ago he was suddenly seized with a rigor, vomiting, and pain in the left side, which confined him to bed for three days. He then went to King's College Hospital, where he was treated as an out-patient for a short time; but he has been quite unable to do his work, and has been very ill ever since. On admission, examination of the chest showed dulness and signs of consolidation over the whole of the upper lobe on the left side, with some evidence of excavation under the clavicle. At the right apex there were signs of commencing consolidation. The patient was confined to bed at first, but was able to be up occasionally for two or three weeks; he is now again confined to bed entirely. During the whole time he has been in the hospital he has suffered from remittent pyrexia. In his case the onset was typically pneumonic; but now, four months after the onset, the disease has somewhat abated in its severity, though the patient is still very much prostrated.

Unfortunately, the victims of acute pulmonary tuberculosis are too ill to be brought down from the wards for examination.

(To be concluded.)

* RENAL TUMOURS: AN ANALYTICAL STUDY.

By J. BLAND SUTTON,
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WHENEVER surgeons invade and effect a conquest on organs previously in the undisputed possession of physicians, the advantage to the scientific side of medicine is always very great. Among the recent advances in abdominal surgery, nephrectomy must be included, and a careful study of the facts, published during the last ten years, throws much valuable light on the characters of tumours of the kidney.

To-day, I shall restrict my remarks to the following genera of tumours:—1, sarcoma; 2, papilloma; 3, adenoma; 4, carcinoma, and will endeavour to analyze their pathological characters

and clinical features in order to ascertain how far it is possible with our present knowledge to answer the following questions:—

1. *Is it possible to foretell the nature of a solid renal tumour from the physical signs?*
2. *Will the removal of the tumour prolong the life of the individual?*

For this purpose it will be necessary to consider each genus separately.

SARCOMATA. As is the case with several organs, e.g., the testis, ovary, and eyeball, the kidney is liable to sarcomata at two distinct periods of life, which not only differ in structure but in their clinical characters.

Infancy. During the first five years of life the kidney is particularly prone to sarcomata.



FIG. 1. A renal sarcoma removed from a boy, æt. 20 months. (Museum, Middlesex Hospital.)

These tumours are usually encapsuled, and in their early stages do not greatly distort the shape of the gland: occasionally a sarcoma will attain the size of a cocoa-nut, and the kidney will be found flattened against it as in fig. 1. Very large tumours will almost entirely destroy the kidney. These sarcomata are composed of round and spindle-shaped cells in which clusters of tubules occur. The majority also contain large spindle-shaped cells possessing a cross striation, so that in sections prepared for the microscope a col-

lection of these cells produces an appearance similar to that of voluntary muscle-fibre. When tubules are very abundant in the tumour the striated spindles are, as a rule, scanty and *vice versa*. When the striated tissue is abundant the tumour is often called a myosarcoma.

Clinical Features. In half the cases, both kidneys are affected: the tumours grow very rapidly, and may attain the size of large melons in a few months: they are absolutely painless: hæmaturia is very rare: secondary nodules form in the lungs on account of processes of the tumour creeping into the renal vein and extending even into the vena cava; fragments from these intravenous processes become detached, and are carried as emboli to the pulmonary vessels.

Death is usually due to mechanical causes:—the tumour, or tumours when bilateral, impede the action of the diaphragm: or processes of the tumour plug the large abdominal veins, and occasionally death occurs suddenly from pulmonary embolus; or in consequence of a piece of growth becoming arrested at the right auriculo-ventricular orifice.

Treatment. The removal of renal sarcomata from infants is a useless proceeding. I have collected the records of twenty-one operations with the following results:—Nine recovered from the operation, and twelve died. The nine which survived nephrectomy died within a year of the operation, from recurrence; half of them were dead within six months. Many of these operations were carried out by surgeons famous for their operative skill, and the records were collected from British, American, French, and German periodical literature. Children under five years of age bear operations on the viscera badly.

Adults. The consideration of renal sarcomata in adults is complicated in an unexpected manner, for it is now clear that certain tumours, thought to be of renal origin, really arise in the adrenals (supra-renal capsules), and some probably originate in "accessory adrenals" embedded in the cortex of the kidney. Therefore in considering supposed renal sarcomata in adults we must distinguish between:—

1. Renal sarcomata.
2. Adrenal tumours.
3. Accessory adrenal tumours.

These distinctions are of first-rate clinical importance, as I will now endeavour to show.

Renal Sarcomata in adults are usually composed

of spindle cells. They are most common between the ages of 30 and 50 years. One kidney only is attacked. The tumour grows rapidly, gives rise to frequent attacks of hæmaturia, and often great local pain. The kidney attains the size of a child's head, and secondary deposits occur in the lungs and liver. Death is due to exhaustion, which is often greatly increased by the anæmia induced by the hæmaturia.

Treatment. As in infancy, nephrectomy in adults for sarcoma is attended with a high mortality, and the duration of life in successful cases is rarely prolonged beyond a year. In adults, however, nephrectomy is occasionally performed to relieve the pain which many of these patients suffer. I collected the records of fourteen cases of nephrectomy for renal sarcomata by British operators: there were six recoveries and eight deaths. Those which survived the operation died within twelve months.*

ADRENAL TUMOURS. The adrenal (supra-renal capsule) may become transformed into a large tumour much in the same way as the thyroid gland becomes a goitre: such tumours closely simulate the clinical features of renal tumours. Like sarcomata of the kidney they occur during infancy, and at this period of life are usually bilateral. Our knowledge of adrenal tumours in children is at present entirely derived from the *post-mortem* room. In the adult, it would seem that they are unilateral, but they may attain a very large size (twenty pounds). In structure they are identical with the zona fasciculata of the adrenal.

Although in situation and from physical signs it is impossible to distinguish between a renal or an adrenal tumour, it has been pointed out that the hæmaturia, which is such a constant sign in renal sarcomata of adults, is absent in tumours of the adrenal.

The surgical treatment of adrenal tumours is very encouraging. Thornton removed two of these tumours, one from a woman 53 years of age which weighed eleven pounds, she was alive and well six years later; the other from a woman 36 years of age which weighed about twenty pounds, this patient was alive and well one year afterwards.

ACCESSORY ADRENAL TUMOURS. It has long been known that accessory adrenals are by no means uncommon; also that certain small yellowish nodules occasionally met with in the cortex im-

* The tables and references to these cases are given in my work on "Tumours."

mediately beneath the capsule of the kidney (often mistaken for fatty tumours of the kidney) exhibit structural characters indistinguishable from the zona fasciculata of the adrenal. The interest in these little bodies has been considerably quickened since it has been observed that large tumours, often confounded with sarcomata, are sometimes found growing in connection with the kidney, as in fig. 2, but being structurally identical with the zona fasciculata of the adrenal. These tumours have a yellowish white colour on section, and exhibit a radiate appearance, the uniformity of which is here and there interrupted by extravasations of blood.



FIG. 2. An accessory adrenal tumour of the kidney. (Removed by Mr. Henry Morris.)

With the limited evidence at my disposal it is difficult to indicate any clinical signs of value in differential diagnosis of these tumours; occasionally they cause hæmaturia, some are the source of great pain, and there is reason to believe they may give rise to secondary deposits.

These tumours are probably very rare, but they deserve attentive study because it will probably turn out that they give the most favourable results after nephrectomy. I am inclined to believe that the case reported by Ris, in which a woman was alive and well five years after removal of a kidney occupied by a so-called *adeno-carcinoma*, the tumours in reality belonged to the species under consideration. So far, all the known cases have occurred in adults over forty-five years of age.

PAPILLOMA. This is a very rare species of

tumour to attack the kidney: it consists of delicate papillomatous processes sprouting from the renal pelvis which are identical in structure with the typical villous tumours of the bladder.

Dr. Murchison described a case in which villous processes occupied the pelvis of both kidneys, and small clusters of similar villi grew around the vesical orifices of the ureters.

The clinical features of renal papillomata may be summarised thus:—They are unknown before the age of fifty, and have been observed as late as the seventy-sixth year. The patients are liable to

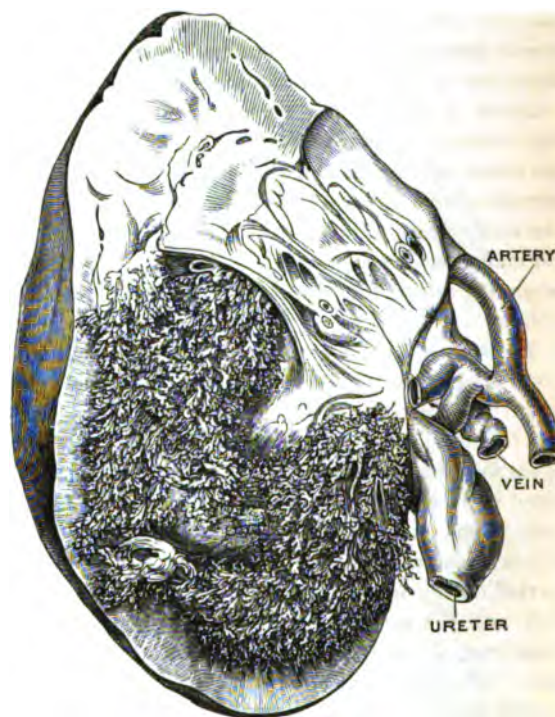


FIG. 3. Papilloma of the renal pelvis. (Nat. size.)

severe and frequent attacks of hæmaturia, accompanied by pain in the region of the affected kidney. When clots of blood pass down the ureter they may produce renal colic. In one case death was attributed to uræmia caused by clots of blood plugging both ureters. In the intervals between the attacks of hæmaturia no pain is felt in the renal region. No case has yet been recorded in which the kidney was enlarged to such an extent by papillomata as to give rise to a tumour clinically appreciable.

Treatment. The only recorded instance known to me in which a renal papillomata has been sub-

jected to operation is a case described by Mr. T. Jones, in which a man, 55 years old, suffered for eighteen months from hæmaturia, accompanied with throbbing pain, in the region of the right kidney. This kidney was explored, and a papilloma was removed from its pelvis. The patient recovered, and the hæmaturia ceased. I have endeavoured, but without success, to learn the subsequent history of this interesting case.

ADENOMA. The kidney tumour which perhaps merits this name, is that known by the phrase "congenital cystic kidney." For a long time I shared the belief in common with many other observers that this curious condition when present in an adult, was, in all probability, of congenital origin, but a careful examination of the excellent series of cystic kidneys in the museum of the Royal College of Surgeons, as well as many specimens which have come under my own observation, have convinced me that the change also arises in adult kidneys.

The general characters of a renal adenoma may be gathered from the specimen sketched in fig. 4.



FIG. 4. A renal adenoma in section. ($\frac{1}{2}$ Nat. size.)
(Museum, Royal College of Surgeons.)

The disease is in the greater number of cases bilateral, but it may be restricted to one kidney. When bilateral, one kidney is almost invariably larger than its fellow; but I have seen both

kidneys of equal size, the shape of, and as big as pumpkins.

The disease begins as minute, scarcely discernible cysts scattered through the cortex of the kidney; these gradually enlarge and destroy the adjacent renal tissue until the gland on section has the appearance of a sponge; by degrees the secreting tissue of the kidney is destroyed, and the gland is merely represented by an encapsuled collection of thin-walled cysts. The ureter retains its normal calibre.

At present no facts are forthcoming to render a diagnosis possible. The patient comes under observation with a swelling in the loin, which has been discovered by chance; it causes no pain or inconvenience. Clinically, the swelling furnishes the physical signs of a renal tumour. When left to itself the tumour increases in size, then the urine assumes the characteristics of granular contracted kidney; uræmia supervenes, and the patient dies.

Bilateral renal adenomata is a very important condition to bear in mind, because, when the disease is so advanced that the kidney is large enough to become appreciable to clinical investigation, it has probably wrought such destructive effects upon the secreting tissue of the kidneys, that the combined efforts of both glands is barely enough for the needs of the individual; a glance at fig. 4 is sufficient to indicate the small amount of available renal tissue. Should this slender amount of "renal capital" be suddenly diminished one half, or perhaps more, by the removal of one kidney, the individual runs immediate risk of "renal insolvency," terminating in death. It is somewhat singular that life may continue for several days in an individual absolutely deprived of renal tissue.

Pathology indicates, and clinical observation serves to confirm the opinion, that nephrectomy for renal adenoma in an advanced stage will rarely be resorted to.

CARCINOMA. It has been so customary to group in an arbitrary manner malignant tumours of the kidney under the name "encephaloid cancer," that it is impossible to give much information in regard to renal cancer. In renal adenomata we have to deal in all probability with aberrations of the uriniferous tubules; and in renal cancer to follow out the precise notions now prevailing in regard to the use of this term, the tumour should be a caricature of the glandular tissue of the kidney. I

have satisfied myself, from an examination of fresh material, that the kidney is liable to malignant tumours, composed of tubules beset with clusters of large epithelial cells, and the excessive development of this epithelium is the predominating histological feature of the tumour. From the scanty knowledge available it appears that renal carcinoma is most common between the fortieth and sixtieth years. It is limited to one kidney and tends to infiltrate it, and invades the pelvis and ureter. Hæmaturia is a constant sign. With



FIG. 5. A renal carcinoma in section: a process of the tumour has invaded the ureter: from a man æt. 45.

regard to the average duration of life, and the tendency to dissemination, no evidence is forthcoming. This may in part be due to the rarity of carcinoma of the kidney, and the infrequency of nephrectomy for this condition. Nevertheless, it is to be expected that in the future, with the spreading interest now surrounding renal surgery, that some complete cases will be recorded. By complete cases I mean a record of the clinical signs, the structural characters of the tumour furnished by a competent pathological histologist, and the subse-

quent course of the case with a description of the *post-mortem* condition of the body, especially in regard to secondary deposits.

My object in insisting on a detailed classification of renal tumours is to demonstrate that the results of indiscriminate removal of these tumours is not reassuring. The operation is attended with a high mortality, and in the majority of cases which recover from the operation life is rarely prolonged beyond a year. It is therefore necessary to subject these tumours to a rigid analysis, clinical and pathological, for the purpose of discovering which tumours may be removed with the best hope of prolonging life, and to obtain, if possible, evidence which will enable us to diagnose, with a reasonable amount of confidence, the nature of the tumour from the physical signs.

Experience at present indicates that the removal of renal sarcomata in infants is a useless proceeding. In adults nephrectomy for sarcomata may be necessary to relieve pain. The removal of adrenal tumours is followed by very encouraging results.

Nephrectomy. The method of removing a solid renal tumour which gives the best results is the reverse of that employed for hydronephrosis. The tumour is exposed through a small incision in the ilio-costal space, and its posterior surface thoroughly detached from the capsule by means of the finger. The patient is then placed in the dorsal position, and an incision made in the linea semilunaris directly over the tumour. The peritoneum is then incised on the outer side of the ascending or descending colon as the case may be, and the kidney enucleated from its capsule. This is a very simple process after its posterior connections have been freed through the incision in the loin. It is of great importance to keep in mind this point, *do not begin to enucleate until the finger enters the true renal capsule*. Non-observance of this will convert a simple into a laborious, tedious and dangerous operation.

Having freed the tumour from its parietal connections, the pedicle is next secured with silk ligatures. In applying the ligature, the pedicle, consisting of the renal artery, vein, and ureter, with the accompanying nerves, lymphatics, fat, and usually a reflection of the capsule, is transfixed as near the tumour as possible. Take no thought of the ureter; if in the process of enucleation the ureter becomes isolated, then secure it with a separate ligature. The ligatures are secured as in

ovariotomy. Do not clamp the pedicle with large forceps and then transfix it; several grievous accidents have happened from this procedure. After securing the pedicle, the tumour is cut away. The abdominal incision is then sutured and the lumbar wound is utilized for drainage, care being taken that the tube enters the old bed of the tumour. The subsequent treatment is conducted as after ovariotomy.

TWO LECTURES

ON

THE USE OF ANTISEPTICS IN MIDWIFERY.

Delivered at the Middlesex Hospital, October 10, 1893,

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LECTURE I.

GENTLEMEN,—The subject which I bring to your notice to-day is one of great practical importance. It should have place in most, if not in all, obstetric manipulations. It forms, therefore, a fitting subject for an introductory lecture in the course of practical midwifery. I shall speak first of the importance of antiseptics in obstetric practice, then indicate the direct objects of each antiseptic procedure, and the best way of carrying it out in practice, and finally discuss the comparative value of various antiseptic agents.

I. THE IMPORTANCE OF ANTISEPTICS IN OBSTETRICS.

Antiseptics are employed in midwifery in order to minimize the risks of blood-poisoning. That this risk is by no means inconsiderable, may be judged from the Registrar-General's mortality return, under the head of puerperal fever, in which, as is on all hands admitted, the deaths returned from this cause falls considerably short of the actual number. Yet taken as it stands, a death-rate exceeding two per 1,000 deliveries, from this cause alone, is registered.

But even this mortality, serious as it is, denotes but a small proportion of the total risk. To the death-rate must be added the danger, from the same cause, of illness, and often of serious and

protracted illness unattended with an immediately fatal result. I know of no figures comparable to the Registrar-General's mortality return which will represent this morbidity on an extensive scale. But this I do know, that gauged by an accurately recorded hospital experience extending over some years (according to which for every death from blood poisoning that took place, some thirty-two cases of illness occurred which were attributable to the same cause), the mortality return must be increased about thirty-fold to represent the amount of illness falling also under the head of puerperal fever, but not immediately fatal. The cases of disease, tubal and chronic pelvic inflammation in one form or another which crowd the out-patient department of our hospitals, may, in the main, be taken as an index of the more protracted of such cases. Collectively, what a tale of misery do they not represent!

But, great as is the risk, a point has now been reached when it is possible to assert that, by suitable measures in which antiseptics pay no inconsiderable part, this risk may be practically avoided.

It is not many years ago that, owing to the ease with which the disease could be spread under the then existing conditions, the mortality and illness from blood poisoning in our lying-in hospitals often reached appalling proportions, and so far exceeded what was then believed to be that of obstetric practice generally, that demands were made, not altogether unreasonably, for the extinction of such institutions. For example, in 1838, of seventy-one women delivered in the General Lying-in Hospital, nineteen died; in 1861, fourteen died out of 195; and in 1877, nine out of sixty-three. Mark that—one death in every seven confinements. It seems almost incredible.

For many years, as may be seen by the accompanying table, the death-rate exceeded 3 per cent.

Death-rate of the General Lying-in Hospital from 1833.

Period.	Deliveries	Deaths	Average death-rate from all causes.
1833-1860	5833	180	1 in 32½ = 3.085%
1861-1877	3773	64	1 in 58½ = 1.696%
1880-1887	2585	16	1 in 161½ = 0.618%
1888-1892	2364	9	1 in 262½ = 0.380%

Between the years 1861 and 1877, before and during which time thousands of pounds were being expended on structural alterations, the mortality

fell to nearly half the previous amount. But even then, at times the mortality was so grave that it was deemed necessary to close the institution, and for three years it remained untenanted. Since 1879, when the same institution was re-opened, the service has been conducted upon aseptic principles. Mark the difference in the mortality then. The value of the adaptation of Listerian measures to obstetric practice is evident from the decline in the death-rate which ensued.

It will be observed that the above table gives the total mortality, not merely the death-rate from puerperal fever. Owing to the defective reports, I am unable to dissociate the septic from the non-septic cases in the early years. But the following table giving the cause of each fatality since the hospital was re-opened, shows at a glance that it is mainly by the elimination of septic cases that

solution and Condyl's Fluid as the general antiseptic employed, with very marked benefit. Since that time, but three deaths have taken place from septic causes among 3,778 patients delivered, and none during the last four years. I may mention, moreover, that these fatalities were the result of dearly-bought experience. One death took place when Sublimate douche solutions of weaker strength were tentatively employed, and the remaining two during the two months in which Salufer douching was tried instead of Sublimate. Apart from these septic cases, twelve deaths have taken place from eclampsia, hæmorrhage, and other accidental complications, such as must inevitably occur in any considerable number of obstetric cases, especially in an institution to which a large proportion of the more serious cases naturally gravitate, and to which several complicated cases were sent

Deaths from Septic and Non-Septic Causes in the General Lying-in Hospital since 1879.

YEAR	1880	1881	1882	1883	1884	1885	1886	1887	1888	1889	1890	1891	1892
DELIVERIES	230	172	325	341	334	395	383	404	497	484	430	463	490
DEATHS—													
From septic causes	(a) 1	—	(d) (e) 2	(g) (h) (i) 3	(j) 1	(k) 1	—	—	(q) (r) 2	—	—	—	—
From non-septic causes ..	(b) (c) 2	—	(f) 1	—	—	(l) (m) (n) 3	(o) 1	(p) 1	—	(s) (t) (u) 4	(w) 1	(x) (y) 2	—
Total deaths	3	0	3	3	1	4	1	1	2	4	1	2	0

- (a) *Septicæmia, abscess in uterine wall.*
- (b) *Pneumonia, syphilitic ulceration of larynx.*
- (c) *Puerperal eclampsia.*
- (d) *Pyometritis, peritonitis, septic phlebitis.*
- (e) *Purulent peritonitis, suppurating pelvic cyst (craniotomy).*
- (f) *Puerperal mania.*
- (g) *General septic infection.*
- (h) *Septic phlebitis, acute endocarditis.*
- (i) *Pelvic abscess, pyolymphangitis, perimetritis.*
- (j) *Pyometritis, septic phlebitis, acute endocarditis.*
- (k) *Septic phlebitis and general pyæmia.*
- (l) *Advanced pulmonary phthisis.*
- (m) *Advanced pulmonary phthisis.*

- (n) *Mercurialism and morphism.*
- (o) *Puerperal eclampsia.*
- (p) *Puerperal eclampsia.*
- (q) *Sloughing of soft parts, parametritic abscess.*
- (r) *Hospital gangrene.*
- (s) *Hæmorrhage (accidental and postpartum).*
- (t) *Accidental hæmorrhage.*
- (u) *Accidental hæmorrhage.*
- (v) *Exhaustion, prolonged labour, chronic nephritis (Cæsarean section).*
- (w) *Exhaustion, prolonged labour, cancer of uterus. (Cæsarean section).*
- (x) *Rupture of uterus, prolonged labour, hydrocephalus.*
- (y) *Syncope, unavoidable hæmorrhage, heart disease.*

improvement has been effected. It will be observed, moreover, that the advantage gained at the outset has since been still further improved upon. This secondary improvement followed upon increased experience in the use of antiseptic measures, and their more perfect application since their first adoption.

For instance, in the early part of 1884, Perchloride of Mercury solution was substituted for Carbolic

after having been days in labour already. But in none of the fatalities classed under this heading has septic infection borne any part. Further, as the mortality from sepsis has decreased, less fatal cases of illness from the same cause have diminished in corresponding degree.*

Nor is this experience of the General Lying-in

* See "Fever in Childbed, Part I., General Hygiene and Antisepsis," in Trans. Obst. Soc., vol. xxxii., 1890.

Hospital at all exceptional. It is the same in all lying-in hospitals and maternities where aseptic principles have prevailed, both at home and abroad. If, in our lying-in hospitals, which at the outset were in an exceptionally bad condition, it is possible, as has been demonstrated, by antiseptic precautions to reduce not only the actual mortality, but also all septic illness to vanishing point, it should be possible to effect this reduction likewise in private practice. That it does hold good in the private practice of individuals I fully concede, but I regret to add that I can show you that at present it is far from being the general case.

On this point, also, I may lay claim to speak with some authority, having recently drawn attention to the subject in the medical press.* But, before discussing that phase of the question, let me also draw your attention to the fact that during last year 1,291 women were attended and nursed in their own homes by the trained midwives of the General Lying-in Hospital, without one single death, thereby affording additional proof of the efficacy of antiseptics to cope with septic disease, even in spite of the indifferent surroundings met with among the poor of the district south of the Thames.

If, then, this result can be achieved in a lying-in hospital, and can be attained also in one class of the community, and that not the better circumstanced, surely it could and ought to prevail in midwifery practice generally. I now invite your attention for a moment to a larger sphere of practice.

Taking the Registrar-General's reports from 1847 to 1892, I find the death-rate of childbirth, calculated for 10,000 confinements, from all causes to have fallen in London from an average of 54.7 before 1860 to 37.4 since 1880; in the provinces for the same periods from 50 to 48.9 only. The death-rate from puerperal fever has, for the same periods and calculated for the same number, fallen in London from 24.1 to 21.5, but in the provinces has increased from 15.8 to 25.6.

Of course, during the 46 years I have taken, there have been many fluctuations. I regret that I am not able to show you the charts illustrating this, but will do so at my next lecture.

The conclusions are obvious. In London, though the total mortality from childbirth has considerably diminished, the improvement has taken place on account of accidents of childbirth alone, the death-rate from puerperal fever

remaining all but stationary. In the provinces, while the total mortality remains at nearly the same level, some improvement has taken place under the head of accidents of childbirth, but the death-rate from puerperal fever has appreciably increased. From this but one inference can be drawn. It is this. No approach towards the general adoption of *efficient* antiseptic measures has yet been made. I say *efficient* advisedly. The main object which I have in view on the present occasion is to indicate how aseptic principles may be efficiently carried out in practice, and to give some reason for their adoption.

A death-roll resulting from preventible disease, comprising 313 deaths in London and even more in proportion in the provinces, returned last year under the head of puerperal fever besides an indefinite amount of illness often producing serious and lasting consequences, offers sufficient indication of the necessity for pressing the matter upon your notice at the outset of your obstetric career.

2. THE TECHNIQUE OF ASEPSIS.

Aseptic principles demand the employment of antiseptic measures. For, surrounded as we are on all sides by various sources of septic contamination, strive to avoid them as we may, antiseptics of one kind or another are required in order to destroy septic matter. But I would not, in order to secure asepsis, for a moment counsel the routine employment in general practice of all such antiseptic measures as are necessitated by the service of a lying-in institution. Though, I may remind you, that even in private practice, circumstances may occasionally arise which require the most elaborate precautions. But, as a rule, the demands of the case are very different. In private, it is possible to cater separately for the needs of individuals, but, in hospital, it is not easy to discriminate; and, therefore, while in both the principle of securing and maintaining aseptic conditions remains unaltered, the measures adopted call for considerable modification.

As I remarked at the outset, the ultimate object of employing antiseptics in midwifery is to minimize the risks of blood-poisoning—in other words, to secure and to maintain a condition of asepsis. But, unfortunately, the means adopted, ostensibly with this end in view, are often little calculated to minimize the risk, and, in fact, may sometimes positively increase it. For the *technique* is somewhat difficult and the pitfalls many. I would

* 'The Mortality of Childbirth,' Lancet, July 1, 1893.

commence, therefore, by asking you, before adopting any antiseptic measure in practice, to consider the more immediate object which it is destined to fulfil; to retain only what is efficient; to reject what is superfluous, and finally to beware of what is dangerous.

Were it possible to regard the parturient tract as being invariably in an aseptic condition at the time of labour, it would, in order to insure immunity to the patient, suffice to guard against the introduction of septic material from without; for the same reason that a test-tube containing sterilized nutrient medium will remain sterile as long as care be exercised to exclude external microbic influences. In that case, the use of an antiseptic in direct contact with the body tissues—in other words the introduction, by means of the douche or otherwise, of any antiseptic into the vagina—would be no more essential than the addition of some antiseptic to nutrient medium in a test-tube.

But, unfortunately, the vagina, even in healthy women, teems with germs and with the products of germs, innocuous for the most part but sometimes noxious. As a familiar example, let me ask you to bear in mind the state of the passage after gonorrhoeal infection. But as a rule, until delivery takes place, with the attendant bruising, and often tearing, of the tissues of the cervix, vagina, and perineum, the germs and germ products, even when toxic, can do no more harm than those which pervade the buccal mucous membrane and external surface of the body; for till then a suitable site is wanting for their passage into the tissues.

But after labour, both the germs themselves and the toxic principles of their production are liable to obtain an entry through wounds into the tissues, and, by so doing, to empoison the body. And, in addition, there is always the risk after delivery that they may travel upwards into the cavity of the uterus, where a large absorbing surface, provided not only by the placental site, but by the entire inner surface of the womb, is for a time always open to them. As a rule, however, in contradistinction to the vagina, the cavity of the womb is not the natural habitat of microbes; even in cases where the foetus dies, for, as is well known, decomposition does not occur, unless the membranes be ruptured or the uterine contents be otherwise disturbed and septic material be introduced from below.

For the reasons detailed above, it is advisable, therefore, to adopt measures in order to sterilize

both the tube and its contents, as well as to guard against the introduction of septic material from without.

We have now arrived at this point—that antiseptic measures, in order to prove effectual, must be so adapted as (1) to secure asepsis by destroying septic material, which may be present in the lower part of the parturient canal: (2) to maintain asepsis by preventing the introduction of septic material from without.

The principle of securing and maintaining asepsis, and thereby shielding the patient from the risks of blood poisoning, is essentially the same in a labour case as in a surgical operation, such as ovariectomy. In order to place the matter before you in a clearer light, I emphasize the fact that the measures adopted with this end in view should be strictly analogous in both instances.

Some time ago, when about to remove a tumour from the pelvis by abdominal section, I asked the patient's own doctor if he would wash his hands and steep them in antiseptic solution in case his assistance were required. He expressed the greatest surprise, saying that he thought that all such measures had been everywhere abandoned, and that it had been shown that they were quite superfluous. I mention this as an example of the erroneous impressions which now and again prevail on the subject of asepsis and the use of antiseptics.

In order to prevent such misunderstanding, let me briefly mention what are usually considered the essential conditions for insuring asepsis in any such operation.

(1) In the first place, before the incision is made, the skin of the abdomen is well washed with soap and water, and subsequently disinfected. This is done, in order to free the field of the operation from possible septic material, lest in any way the surface of the body may have been contaminated.

(2) From the beginning of the operation until the wound is closed, the greatest care is exercised that the hands, instruments, sponges, ligatures, and in fact, everything brought into contact with the wound, or passed through the wound into the abdominal cavity, should be previously rendered aseptic. This is done, in order to free what might otherwise serve as vehicles of infection from possible septic contamination.

(3) And, finally, that the dressings, or at any rate, such of them as are in contact with the

wound, should be aseptic, and so maintained until the wound has healed. This is done, with a view to protect the wound from the possibility of septic contamination after the operation has been completed.

In addition to these, there are many subsidiary points, some of a general, others of a local significance, which are of great importance in themselves, and on that account alone demand careful attention; but which, if aseptic conditions be not otherwise secured, may also be of importance in controlling and combatting the development of sepsis. Such are—maintaining the general health of the patient; securing to her healthy surroundings, rest both mental and physical, fresh air and nutritious food; guarding against infection from zymotic and other febrile diseases; regulating the eliminatory organs, particularly the bowels and kidneys: and, locally, expediting the operation; controlling hæmorrhage; avoiding unnecessary examination and manipulation; limiting the injury done to the tissues to the smallest possible amount; removing as far as possible all blood clot and tissue deprived of vitality, and accurately adapting the edges of the incision wound. By attention to these points distinct advantage will be secured.

The counterpart of these matters in obstetric practice is equally important. And, lest it be thought that by dismissing them with casual mention they are to be considered as trivial or of no importance, I feel bound, at any rate, to enumerate them.

The subsidiary points, which are of a general character, are the same, and their importance will doubtless commend itself to all, whether applied to a capital surgical operation or to a labour case, for anything which tends to lower the general vitality of the patient places her in a less favourable position to withstand the onslaught of septic mischief. The innate power of resistance in healthy tissues is a factor of such importance that we cannot afford to disregard it, lest we expose the patient to additional risk by diminishing it. It is an undoubted fact that, whether leucocytes be the active agents in the process or not, the tissues of the body generally offer an active resistance to septic poisons, and up to a certain point are, without extraneous aid, capable of annihilating and destroying them. It is only after this point is passed, that the ill effect of the poison can assert itself. Any cause, therefore, which tends to lower the general health, will obviously diminish this resisting power, and place the patient at a disadvantage.

All such measures, whether local or general, as are capable of expediting the process of labour and of preventing unnecessary expenditure of energy on the part of the patient under various conditions—normal and abnormal—and of preventing undue loss of blood, also tend in the same direction. These matters I shall have occasion to discuss with you in detail later in the session. For the present I will rest content with pointing out that all unnecessary examinations and manipulations should be carefully avoided, because each in itself provides a possible means of conveying infection and of inflicting mechanical injury on the tissues locally. When examinations are made or operations in cases of necessity undertaken, care should be exercised to limit the injury done to the smallest possible amount, so as neither to deprive the tissues of their vitality, nor to diminish the power, which they naturally possess, of destroying septic material which may perchance reach them, nor finally to provide additional sites for absorption.

The retention of blood clot and of portions of the placenta and membranes should also as far as possible be guarded against, not only as entailing the risk of hæmorrhage, but because, in the first place, being deprived of vitality, retained products are capable of providing a suitable nidus for the development of septic organisms, and often form, as it were, a ladder for their passage from the vagina into the uterus; and, in the second, because by their presence they retard involution and keep the surfaces in a state capable of absorption.

Finally, by accurately adjusting the edges of wounds about the vulva and perineum, and immediately closing such lacerations by suture, the area for absorption is diminished in the situation where of all others asepticism is liable to fail.

But these, as I have already remarked, are subsidiary points, and though of such importance as to demand mention, do not immediately concern the special subject of this lecture.

ESSENTIAL CONDITIONS TO BE FULFILLED.

We have now to consider the conditions which may be regarded as essential in order to insure asepsis in a labour case.

(1) In the first place, before delivery takes place, and absorbing surfaces are laid bare, the vagina should be flushed out, and as far as possible disinfected, and the external genitals washed and similarly treated. This is done in order to free the field from possible septic material.

(2) From the time of delivery until external lacerations have healed and the lochia have ceased, scrupulous care should be exercised that the hands, instruments, and in fact everything brought into contact with the vulva or passed into the vagina or uterus, and so brought into touch with absorbing surfaces, should be aseptic. This is done to prevent the introduction of septic material from extraneous sources.

(3) And, finally, care should be taken that the pad, or at any rate such part of it as is brought into contact with the vulva, should be aseptic, and so maintained until the wounds shall have healed and the lochia ceased. This is done with a view to prevent the possibility of subsequent septic contamination.

It remains to consider by what special means the objects above enumerated may be best carried out in practice.

To wash and disinfect the skin of the abdomen and to render aseptic the field of the operation in a case of ovariectomy, is a comparatively easy matter. But it is far otherwise with the vagina which is much less accessible than the flat surface of the abdomen. And, owing to the rugose condition of the vaginal mucosa, and the presence of tenaceous mucus, it has proved an exceedingly difficult matter to render the canal aseptic, even when by the aid of a speculum the folds have been opened out, the mucus mopped away with cotton wool, and an antiseptic solution brought directly into contact with the mucous membrane. Anyone who has treated gonorrhoea in this way will appreciate the difficulty. Such being the case, it is even more difficult to effect the same purpose by merely syringing the vagina with germicide solutions.

But, by first washing away the mucus by a full stream of warm water, and then irrigating the vagina with two quarts or more of solution endowed with strongly antiseptic properties, a great deal may be done to rid the canal of septic material, and to remove at least so much as lies free within it. If no more than that be effected, considerable gain will have been achieved, for what the douche fails to remove or to destroy is not likely to be carried higher up into the canal during the examinations and manipulations incidental to all ordinary labour. And, immediately after delivery is completed, the very best opportunity is afforded of ridding the vagina from any septic material which may remain. During the course of labour the flushing out process has in

some measure been continued naturally by the flow of liquor amnii, and also in the separation of the placenta by the flow of blood, while during the second stage the folds of the vagina have been more efficiently opened out by the passage of the foetus than could be done by any artificial method. At the completion of delivery, the douche is usually called into requisition for other purposes—by flushing out the passage, to aid the removal of detached portions of tissue and of blood clot, or given at a temperature of 110° – 115° F., by stimulating the uterine muscle to action, to prevent the formation and assisting the expulsion of retained products. As, at this stage, it is possible to effect this additional object with but little additional trouble, it is better to combine an antiseptic with the douche. But in employing the douche, especially during puerperium, care should be exercised to wash first the parts about the vulva and the external genitals; then to douche the vagina, and, only after this has been done, should the tube be carried (and then only when there is some special reason for it) into the uterus itself. By following out this method of procedure, the risk of carrying septic matter from the vulva or vagina into the uterine cavity is reduced to a minimum. Keeping the patient meanwhile in a supine position, the uterus, with one hand placed like a cap, over the fundus, may be supervised and, by now and again compressing and depressing the womb by pressure through the abdominal wall, any fluid which (even when the uterine tube is not used) may have found its way into the uterus will be expelled and clot or retained membrane will be washed out with the gush of water as it escapes from the vagina.

For the purpose of washing and disinfecting the vagina, some form of apparatus is requisite, because the canal is not accessible to the more ordinary cleansing and disinfecting process applicable to the surface of the body. But, whatever form of apparatus be used, I must impress upon you that there is nothing special in it. As a means towards an end some apparatus is necessitated by the special requirements of the case. But it must not be forgotten that, whether applied to the surface of the body or to the vagina, the water and the disinfectant are the important factors.

There is an obvious mechanical advantage, however, in having at command the forcible stream of the continuous douche. This may be obtained not only by means of the usual hydrostatic ap-

paratus, which is barely portable, but, by requisitioning an ordinary ewer and a length of elastic tubing, an apparatus may be improvised which is capable of meeting all the requirements. The tubing can be converted into a syphon by filling the tube with or without the aid of a small glass funnel, which, if attached, has the additional advantage of serving to keep the tube in place; or, by affixing the tubing to an ordinary elastic enema syringe and working the apparatus till the stream begins to flow, a continuous syphon douche may be produced. According to the height of the reservoir above the patient the force of the current can be varied at will.

Owing to the readiness with which it may be cleansed and kept clean, a nozzle made of glass or celluloid is advisable, and for intrauterine irrigation a double channelled tube is preferable.

You thus see how simple and portable the apparatus may be without detracting from its practical value. Moreover, the difficulties of a receiver for the solution as it pours away, in the absence of a bed-bath, may be conveniently overcome by pinning together the two corners of the macintosh hanging over the side of the bed, so as to form an improvised funnel, which carries the solution into a receptacle placed on the floor beneath.

For the purpose of washing and disinfecting the external genitals pledgets of absorbent cotton are to be recommended in place of sponges. Sponges, through constant saturation with blood and discharges, are with difficulty cleansed. At any rate they are not to be trusted; the pledgets of cotton are inexpensive and can be burnt after use.

By observing the above-mentioned precautions during the course of labour, a reasonable hope may be entertained that, when the labour is over, the canal will have been freed from any septic material which it might have contained, and that, unless reinfected by septic material introduced from without, an aseptic condition will be preserved.

It has been urged that, if at the beginning of labour the canal is to be regarded as already impregnated with septic matter, it is useless disinfecting hands, instruments, etc. But at best this is but a poor argument, for, even if one source of infection were invariably present (which is doubtful) in the vagina itself, nevertheless, no inconsiderable gain would be effected by adopting means to eliminate the hundred and one others of extraneous origin which might be conveyed, unless preventive

measures be taken. Still less does this argument hold good when means, such as I have above described, are adopted to disinfect the canal.

And it must be remembered that in any examination or manipulation the finger or instrument, and with it any septic matter adhering, is almost invariably carried to the upper part of the canal, where septic matter is naturally scarce or entirely wanting. I would say then that every reason exists under any circumstances for disinfecting the hands, instruments, etc., employed with as much care as in an ovariectomy operation, whether means be taken before the operation is commenced to disinfect the abdominal parieties or not.

The measures employed to disinfect the hands, instruments, etc., should be identical in the two cases.

At the outset the hands should be well washed with soap and water (special attention being paid to the nails), and having been rinsed, should be immersed for at least a minute in strong antiseptic solution. They should be again steeped in the antiseptic solution *immediately* before any vaginal examination is made or manipulation undertaken. I say *immediately* advisedly. There are obvious risks if the disinfection be performed some time previously, that infection may be picked up in the interval. I need scarcely add that after the examination the hands should be washed to remove the discharges. I may here point out that as either a little soap or a little blood will suffice to throw down Mercury from Sublimate solution, the necessity for this procedure is rendered obvious.

As regards instruments, it is almost superfluous also to say that they should never be put away in a dirty state, but should invariably be cleansed immediately after use. Instruments of bright metal, *e.g.*, the forceps (which are liable to be acted upon injuriously by strong antiseptic solutions), should be kept bright by rubbing, and should be disinfected immediately before use by steeping in boiling water. Others, such as gum elastic catheters, celluloid and india-rubber tubes and syringes, which would be injured by the high temperature, after having been washed and rinsed in water, should be soaked in strong antiseptic solution. Ligatures should be treated by steeping in boiling water immediately before use, and sponges should be steeped in the same way.

These measures as applied to the hands, instruments, etc., demand no special apparatus, but are, nevertheless, of the most vital importance to asepticism.

The pad occupies the same relation to the labour case as the dressings to ovariotomy, and like the pledget of cotton in the test tube to which I previously alluded, is intended to obviate the entry of micro-organisms from without. Now, in order to prevent the access of microbes, which are ever present and are wafted about in the atmosphere to the nutrient medium within the tube, the crudest form of cotton is found to be sufficient, that is, provided the cotton be required to act solely as an efficient filter to the air as it passes in. But if the cotton become saturated it may be otherwise. In that case, it is necessary to renew the pledgets before the microbes reach the portion saturated, otherwise, being deprived of filtering power, the plug becomes no longer capable of preventing the microbes from spreading to the rest of the tube contents. Under such circumstances, advantage would accrue from combining with the plug an antiseptic agent to destroy the microbes. But the same end would be gained by constantly renewing the plug before it became saturated.

Owing to the discharges incidental to a labour case, the pad must inevitably soon become soaked through, unless renewed, and, owing to the close proximity of anus and urethra, the dressing is apt to become soiled and saturated and to require frequent renewal. The same may happen also in an ovariotomy where there is much discharge, and drainage is employed.

Pads of absorbent cotton, or of wood wool enclosed in gauze or lint should be used in place of diapers, and burnt immediately after use, the cost being no greater than the washing of the diapers. I bring to your notice the kind of pad in use at the General Lying-in Hospital. These pads are of the simplest possible construction, consisting merely of a handful of wood wool wrapped in a square of gauze twisted together at the ends in order to retain it in place. The material may be impregnated with some antiseptic, though this adds materially to the expense, and, if they be changed frequently, is not requisite. Certainly the clean dry pad is an additional source of comfort to the patient. It is scarcely necessary to add that in changing the pad, as in renewing the dressings after ovariotomy, any excess of discharge should be removed before the clean dressing is applied.

The special means by which an aseptic condition of the parturient canal may be attained

and maintained have been considered, but I have purposely reserved for separate mention the precise form of antiseptic agent which should be employed, the strength of the solutions, and the most convenient method of preparing them. This will form the subject of the next lecture.*

REVIEW.

Annual of the Universal Medical Sciences.

(Five Volumes.) Edited by CHARLES E.

SAJOUS, M.D. (F. Rebman, London.)

Published at £3 5s.

This forms the sixth issue of what must be regarded as the most complete and exhaustive work of reference to the medical literature of the whole world, being an admirable digest of all recent medical knowledge. It has on its list of reference some 2,000 journals and a large number of books, monographs, theses, etc. To glean important matter from this enormous mass of information, Dr. Sajous has to assist him seventy associate editors and over 200 corresponding editors. The result of their joint labours is presented in five volumes. Each volume is divided into sections; thus, in Vol. I. there are twelve sections devoted respectively to Diseases of the Lungs and Pleura, Diseases of the Heart and Blood Vessels, and so on. Each section is under the charge of one or more assistant-editors. Each volume contains an index to its contents. They are well printed and illustrated, and may be said to bristle throughout with references. The work of extracting from different journals is well done in all sections. In addition to the more general subjects, there are sections devoted to Electro-therapeutics, Climatology, Legal Medicine, Physiology, and many other subjects not, as a rule, touched in the smaller works of the same scope. The index at the end of Vol. V. is divided into three sections, subjects, therapeutics, and authors quoted. We can say nothing greater in praise of it than that it is well worthy of the book. The part devoted to therapeutics is to those who wish to be acquainted with the most recent treatment of disease, invaluable; we can call to mind nothing so exhaustive and complete, arranged in this manner. The names of the different diseases are arranged in alphabetical order, and against each one is placed in brief, various recently-introduced modes of treatment, and the reference to it in the particular volume where the subject is more fully discussed. The Editor is to be congratulated on having secured for this issue the valuable co-operation of such men as Sir Benjamin Ward Richardson, Dr. Dujardin-Beaumetz, of Paris, and other distinguished European physicians.

It is unfortunately impossible to deal at length with such a large work as this. It would be easy to occupy the whole of our issue in discussing this mass of digested practical information. Those engaged in any branch of professional work will find it of value, and those following general practice would find it of equal value, as it includes such varied information in its exhaustive pages. We cordially congratulate Dr. Sajous and his fellow-workers on the success of their efforts to place before the profession so useful a digest of the world's yearly work in medical science.

* This Lecture will be published at the end of this month.

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 8, 1893.

TWO CLINICAL LECTURES ON TYPES OF PULMONARY PHTHISIS IN ADULTS.

Delivered in connection with the London Post-Graduate Course at the Brompton Hospital for Consumption and Diseases of the Chest,

By **PEROY KIDD, M.D., F.R.O.P.,**
Physician to the Hospital; Assistant-Physician and Pathologist to the London Hospital.

(Concluded from p. 4.)

CHRONIC FORMS.

I shall now show you patients exhibiting some of the common types of chronic phthisis, giving a short abstract or outline of each case, which you can fill in for yourselves from your own examination, and from a perusal of the detailed clinical notes which the patients bring with them. In all of these cases the physical signs are most marked at the apex, though in their grouping they present certain differences in each individual.

S., a boy *æt.* 15, has suffered from cough and general symptoms of phthisis for about eighteen months. Physical examination shows signs of consolidation of the left apex, and also of the apex of the lower lobe on the same side, that is to say, at the infra-spinous fossa. This patient is able to walk in the garden, but suffers from a moderate degree of remittent pyrexia.

L., a man *æt.* 25, has suffered from cough for ten months. Nine weeks ago he spat up a large quantity of blood,—two pints,—according to his own account. The physical signs are very slight, namely, scanty subcrepitant râles at both extreme apices, without any dulness or distinct alteration of the breath sounds. This patient has also lost flesh, and has remittent pyrexia. Judged from the physical signs the lesion would seem to be in an early stage; but, from the fact that he has had a profuse hæmoptysis, we may assume that there is much more disease than physical examination discloses,—in all probability a cavity or cavities.

C., a man *æt.* 36, has had winter cough for three years; a continued cough, debility and dyspnoea for the last two months. The physical

signs indicate infiltration of the left apex, and a dry rub is to be heard in the left axilla. The localization of this pleurisy suggests the existence of disease of the lower lobe as well as of the apex of the upper lobe, although there are no further signs of disease of this part.

R., a man *æt.* 31, gives a history of having had cough and expectoration for ten months without much failure of general health. This patient has no marked pyrexia, and does not seem at all severely ill. Physical examination revealed only a slight retraction at the left apex with deficient movement, weak breath sounds, and a shade of dulness above the clavicle and at the supra-spinous fossa. There were no râles. Examination of the sputum showed the presence of numerous tubercle bacilli. This patient has gained weight greatly during his stay at the hospital, and we may assume that in him the disease is fairly quiescent. Though the physical signs are so very slight yet the presence of tubercle bacilli in large numbers in the sputum makes it very probable that he has a small cavity at some point, probably in the left apex.

M., a boy *æt.* 15, tells us that two years ago he had influenza, and has been very weak ever since. Twelve months ago cough developed, and last September he had an attack of "pleurisy." Physical examination showed slight dulness at the right apex with other signs of consolidation, and some scattered râles over both upper lobes on quiet respiration. On making the patient cough a shower of crackles could be heard over the whole of both upper lobes. From this we may conclude that in addition to a considerable area of consolidation at the apex, there are scattered tubercular nodules in both upper lobes, the explosive râles produced by cough being explained, as Dr. Reginald Thompson has suggested, by the sudden separation of the moist surfaces of the catarrhal bronchioles by the forced inflation of the upper lobe which occurs during the act of coughing.

B., a man *æt.* 42, gives a history of cough and failure of general health following an attack of influenza three years ago. Examination of the chest shows contraction of the left side above with signs of excavation under the clavicle, the heart

being uncovered owing to retraction of the anterior margin of the upper lobe of the lung. A short systolic murmur is heard close to the sternum in the second intercostal space. At the apex of the right lung there are slight signs of consolidation. In this patient contraction of the left lung has probably produced slight pressure on the right pulmonary artery or conus arteriosus of the right ventricle, giving rise to the systolic murmur.

Annie P., 32, gives a history of long-continued cough. The patient has some small scars about the mouth and other signs of old syphilis. Physical examination of the chest shows marked contraction of the left side above with displacement of the heart outwards, signs of excavation being present at the apex of this lung. The resonance of the right lung extends beyond the left edge of the sternum, and at the apex of this lung there are slight signs of consolidation. Here the contraction of the left lung has caused displacement of the heart, flattening of the chest wall, and secondary distension of the opposite lung which may in time go on to emphysema.

The cases I have just brought before you are all of the common character which you may meet with any day. Now I wish to show you some of the less common varieties of pulmonary tuberculosis.

The Emphysematous Type.—Charles W., æt. 35, has suffered for ten years from winter cough, which has been increasing in severity for the last few years. The cough has been persistent since last winter, and has been accompanied by wasting and occasional attacks of spasmodic dyspnoea. The patient is fairly well made, but somewhat wasted. Physical examination revealed marked hyper-resonance, and very weak breath sounds, especially on the right side, and scanty subcrepitant râles round the right nipple and at the posterior base. The diagnosis here was emphysema, chronic bronchitis, and (?) tuberculosis. The sputum was examined, and tubercle bacilli were found to be present. You may ask why the presence of tuberculosis was suspected. In answer to this it must be said that the wasting of the patient was the point that specially aroused our suspicions. In another patient, who presents the same physical signs exactly, a history of a large hæmoptysis suggested the same suspicion, which was also proved to be well-founded by an examination of the sputum. In general it may be said that where a patient presents signs of emphysema without the

characteristic conformation of chest which we associate with this condition, and especially if there should be a history of hæmoptysis, or marked emaciation, the question of tuberculosis should always receive most careful consideration.

I may say that in these cases the prognosis is nearly always good as to duration. This form of tuberculosis generally runs a very chronic course.

Pleuritic Variety.—Alfred C., æt. 35, somewhat addicted to alcohol. The patient was quite well until Easter, 1893, when he caught cold. A week later his breath became short, and he gave up work; but he made a partial recovery, and was able to do a little work a few weeks later though his breath was still short. There has been no pain at any time, but he has lost much flesh. Seven weeks ago increasing dyspnoea compelled him to give up work. He was treated at home by a medical man who tapped him three times. On each occasion clear fluid was withdrawn. On admission at the end of last August the patient was seen to be fairly well nourished, and was able to be up and about. On physical examination we found on the right side marked dulness from the third space to the base, weak breath sounds, and diminished tactile fremitus; behind, at the supra-spinous fossa, resonance tympanitic; from the mid-scapular region downwards, marked dulness; very weak breath sounds close to the spine; towards the lateral region, breath sounds absent; tactile fremitus diminished generally. On the left side a small patch of dulness was discovered in the axilla just outside the cardiac area. The heart's apex beat was in the fifth intercostal space about half an inch outside the nipple line: sounds clear. The liver was much enlarged, very firm, and slightly uneven. The diagnosis was pleurisy with some effusion,—(?) tubercular,—and cirrhosis of the liver. Tubercle bacilli were found in the sputum. Paracentesis was performed, and six ounces of clear fluid removed from the right pleura. The patient has suffered all along from remittent pyrexia and has not manifested any improvement. About a month after admission a dry rub was heard in the left axilla, and dulness gradually appeared at the left base. The history of marked wasting, and the existence of double pleurisy made us suspect the presence of tuberculosis, a view which was confirmed by the discovery of tubercle bacilli in the sputum. Pleurisy occurring on both sides, in the absence of renal disease or acute rheumatism, is almost invariably tuberculous. The prognosis in

this class of case, so far as my experience goes, is unfavourable.

ATYPICAL LOCALIZATION OF TYPICAL SIGNS.

Basic Type.—Mary P., æt. 17, was quite well till she had typhoid fever ten years ago, since which time she has been ailing. For four years she has suffered from winter cough. This summer the cough has persisted, and she has suffered from progressive weakness. A short time ago she had a small hæmoptysis. The patient is tall and of a slender build; her fingers are slightly clubbed. Physical examination discloses moderate dulness over the lower third of the left lung posteriorly, with weak tubular breath sounds, bronchophony, and crackling râles, some small and some medium-sized. At the right posterior base a few muffled râles are to be heard. There are no signs of disease in the upper part of either lung. The diagnosis in this case was pulmonary tuberculosis, and subsequently tubercle bacilli were found in the sputum.

Martha M., æt. 19, has for one year been losing colour, and suffering from weakness and dyspnoea on exertion. She has had a very slight cough for the last two months, and has lost a good deal of flesh. She is a tall, slender, and very anæmic girl. At both apices the expiration is prolonged, and there are scanty subcrepitant râles, but no dulness. On the right side posteriorly there is marked dulness over the lower half, and intense cavernous breathing at and around the angle of the scapula, together with pectoriloquy and some medium-sized crackling râles. The diagnosis was phthisis, nodular infiltration of the upper lobes and massive consolidation at the right lower lobe. Tubercle bacilli were found in the sputum.

In the first of these two cases physical signs of consolidation were confined to the base, whereas, in the second case, although the physical signs were most marked at the base, there was evidence of disease at the apices also. May we conclude that in these cases the disease commenced at, or was confined to the base? Certainly not. Some years ago (1886), in a paper on basic tuberculosis published in the "Lancet," I described two cases of primary tuberculosis of the base. At the same time attention was drawn to other cases, in which the diagnosis of basic tuberculosis made during life was shown to be incorrect on post-mortem examination. In some of these there had been physical signs of disease confined to one or both bases, and

yet when the patient died inspection showed that the oldest disease was situated at the apex of the upper lobe. In another group of cases, where slight physical signs had been present at the apices but had preponderated at the base, the necropsy demonstrated the fact that the oldest disease was situated at the apex. In other words, we are not in a position to diagnose primary basic tuberculosis with complete certainty, even when the physical signs are at first confined to the base. This depends on the fact that considerable masses of tuberculous disease or even cavities may exist towards the central part of the upper lobe without giving rise to any abnormal physical signs, owing to the lesions being covered by a shell of healthy lung. Disease confined to the base of one lung in most cases is not tuberculous, and we have in such instances to exclude various diseases which I can merely enumerate:—chronic pneumonia or cirrhosis, with or without bronchial dilatation; localized pleurisy; (on the right side) abscess of the liver projecting upwards and opening into the right lung; new growths; and hydatids of the lung or liver. I shall not attempt to go into the diagnosis of these affections, but will only say that a careful and repeated examination of the sputum will almost invariably decide whether the disease is or is not tuberculous.

Dr. Pollock considers that in the basic type of phthisis prognosis is favourable as to duration; but the cases are really so rare that it is difficult to formulate any definite opinion. Dr. Pollock's views are founded on the clinical evidence of the disease being basic, that is to say, the physical signs were confined to, or most marked at, the base; but, as I have just shown, the clinical method is open to certain fallacies, and cannot be relied upon to settle this question. Among some 900 autopsies on cases of pulmonary tuberculosis in adults I only met with three instances in which the disease commenced at the base.

Physical Signs of Consolidation confined to, or most marked at, the lower part of the upper lobe.—

I have to thank my colleague, Dr. Reginald Thompson, for kindly permitting me to bring this case before you. Charlotte O—, æt. 26, gives a chronic history of cough, expectoration and pain in the left side. On admission, the physical signs were limited to the left side, great dulness being found from the level of the third to the sixth ribs in front, extending from the cardiac region outwards as far as the anterior axillary fold. Over

this area there was marked dulness, weak tubular breathing, and scanty subcrepitant râles. Later on some râles were heard beneath the left clavicle, and they are now very abundant in this position. Tubercle bacilli were found in the sputum. In this case the physical signs indicated a massive consolidation of the lower anterior part of the left upper lobe. Although no evidence of disease of the apex was discovered it is probable that mischief, nevertheless, existed there. In fact, in this type of case it will generally be found, if a necropsy be obtained, that a cavity exists at the apex, the massive consolidation of the lower part of the upper lobe being probably explained by the very active inspiratory expansion of this part of the lung favouring the inhalation of infective material from the cavity above. A similar condition of things is less frequently found on the right side involving the anterior inferior part of the upper lobe or the middle lobe.

Phthisis associated with morbus cordis.—Edward L., æt. 41, had rheumatic fever seventeen years ago, and since that time has had a slight cough, but has been fairly well and has been able to work till twelve months ago, when he was obliged to give up work in consequence of increasing cough, expectoration, and loss of flesh. Physical examination shows slight dulness at both apices, with rather scanty, medium-sized, crackling râles. At the right apex the breath sounds are weak, but of a whiffing, tubular character. At the left apex the expiration is prolonged. The heart's apex beat is in the fifth intercostal space in the nipple line. There is no increase of dulness upwards or to the right. A harsh systolic murmur is heard all over the præcordia, replacing the first sound entirely, loudest at the apex, and conveyed to the back. The pulmonary second sound is accentuated. This patient has double apical tubercular infiltration and chronic rheumatic endocarditis, causing incompetence of the mitral valve.

The prognosis in cases of phthisis associated with morbus cordis is always good as to duration, except in the rare cases in which the endocarditis takes on the malignant form. It is well known that the association of these two diseases is uncommon. Rokitsansky, indeed, went so far as to say that morbus cordis provided a complete immunity against pulmonary tuberculosis. This fact was attributed by him to hyper-venosity of the blood, and the same explanation was invoked by him for the retarding influence of emphysema on

the course of pulmonary tuberculosis noticed by Laennec. Doubt has been thrown upon this explanation, mainly on account of the fact that patients with congenital pulmonary stenosis are very liable to contract tuberculosis; but it is doubtful whether this objection is so fundamental as some have imagined. I cannot go into this question on the present occasion, but I would refer those who are interested in the matter to a paper published in the St. Bartholomew's Hospital Reports for 1887, in which this question is considered.

I do not venture to say that I have shown you all the clinical types that pulmonary phthisis assumes, but you have seen to-day the principal varieties of the disease. An examination of the cases will convince you that phthisis has many atypical manifestations which it is very important to recognize, inasmuch as a due appreciation of this fact has a very direct bearing, not only on diagnosis, but also on our prognosis.

A CLINICAL LECTURE

ON

A CASE OF NECROSIS OF THE FEMUR.

Delivered at Guy's Hospital, October 18, 1893,

By H. G. HOWSE, M.S., F.R.O.S.,

Surgeon to the Hospital.

I HAVE rather an interesting bone case to-day, Gentlemen, and I think I could not do better than improve the occasion by speaking of it. It is a case—but not quite an ordinary case—of necrosis of the femur; and there are such features about it that I think it may very well form the subject of a clinical lecture. I shall give you briefly the history of the patient, and then I can point out to you the various peculiarities the case presents.

The patient is aged 41, an engineer, living in London. There is nothing in the family history of much importance with reference to the patient's case. His mother died of cancer, his father of heart disease; he has no brothers or sisters living, but, on the whole, the family was a healthy one. The patient is single. When a boy he had rheumatic fever. I do not know whether that is a matter of great importance, but still we can record it. Beyond this and children's diseases he knows of no other illness except influenza, which he had

last March, and an attack of syphilis twenty-five years ago; this may have some bearing on the case.

A little over thirty years ago the patient fell down a hole, twenty feet deep, on to big stones, and broke his left femur near its distal end. He was taken into Hull hospital, and during his stay there several abscesses formed, which were cut down upon and pus evacuated. The surgeons wanted him to have his leg amputated, so that his injury at that time must have been a severe one, probably a comminuted, not a simple, fracture. It was sufficiently great, at any rate, to suggest to the surgeons at the hospital the advisability of having the leg amputated. His father, however, would not allow it, and he was sent out of the hospital on crutches, having been in for about eighteen months. He was then attended for eight or nine months by a lady, a herbalist, whose treatment seems to have been fairly successful, whether by the herbalistic treatment or the lapse of time I cannot say (probable the latter), for after four months of it he went to work, only using a stick for support. Clearly, then, there was a considerable improvement in his condition during that time.

There is a considerable interval in the history at this period; and I do not quite clearly know what was the condition of the part during the succeeding years, but I suppose that the abscesses had more or less closed up, and that the patient, although having a disabled leg, was fairly able to earn his livelihood, at any rate to follow his occupation to a certain extent. Nevertheless, about six or seven years ago an "ulcerative rash," as he calls it, whatever that may mean, broke out just at the top of the popliteal space. The patient went to one of our metropolitan hospitals, and was there operated on by a surgeon for necrosis. I think it is pretty clear that at this time there must have been sinuses in connection with the femur through which dead bone could be felt, and which then gave rise to the impression that there probably was some central necrosis in connection with the femur. At this operation much loose bone was removed. He was in hospital for thirteen weeks. There must therefore have been a cavity of considerable size which took a long time in partially closing up. The patient, however, says that he did not get quite well. "He has never been right since," is the expression of the report. From that we may conclude that the disease never wholly disappeared. He has had several discharges of pus at the place, small pieces of bone coming away

at the same time. This is important when taken in connection with the fact that a large quantity of loose bone was removed at the time of operation. He was able to go to work, to a certain extent, for the next three or four years. In October, 1892, however, he went into St. George's Infirmary, Fulham Road, where again the surgeons wanted to amputate the leg. Although he was there for six months he did not care about this being done. On October 10th he applied for admission here.

I have made you acquainted, so far as I know it myself, with the previous history of the patient; and at first sight, to those of you who know anything of such cases, this will seem one of central necrosis, resulting from acute septic periostitis, first started after the fracture to the bone. To a certain extent this is true, but we shall see that there are distinguishing characters in this case, which separate it from an ordinary one of central necrosis. Before, however, speaking of that, I shall give you the condition on admission.

On admission the patient had a swelling round the femur, above the left knee. The report states that the place where the bone was broken could be seen and felt. There was an irregularity here, no doubt; but whether that was due entirely to the fracture of the femur may be subject to question, especially when we remember the very great changes the bone must have undergone in the processes of repair after the operation a few years ago. There were two drainage tubes in two different sinuses on the back and inside of the lower part of the thigh, close by the popliteal space. From both of these bare bone could be felt with the probe. As a measure of precaution, the urine was examined, and nothing wrong as regards it was found—no albumen, no sugar. Its spec. grav. was 1.021.

On the 13th, as we wished to examine him under chloroform, we had him taken into the theatre, and we then found the condition of things mentioned above, viz., that there was a swelling of the lower six or seven inches of the femur, very great thickening of bone, and two or three sinuses which went to the popliteal space, and through which one could feel a large extent of bare bone. These sinuses were discharging an intensely foetid pus—foetid in spite of a previous preparation of the patient by antiseptic washing out of the sinuses. This pus, however, was nothing like so foetid as when the patient was first examined in the ward. It was then so repulsive that it was extremely

unpleasant to remain near him. The washing out with antiseptic lotions had failed to make the sinuses anything like aseptic. The large area of bare bone felt by the probes did not seem to be loose; it appeared to correspond very much with the whole of the posterior aspect of the femur.

The question was, what ought we to do with the patient in this condition? Now, I have very little doubt that the majority among you, looking at a case like this, would say, Here is a patient with necrosis affecting the lower six or seven inches of the femur; why not cut down upon and remove the sequestrum, and then see whether it will not granulate up and soon get better? That, however, when I examined it, was not my view, and, without giving you any definite reasons in the theatre, I told you it would be the wisest course to amputate, thereby agreeing with those who had seen him in St. George's Infirmary. So we prepared to amputate.

I shall tell you now what were the reasons which influenced me in arriving at this determination. It was not, I may say at once, the previous surgical opinions given in this case. I kept my mind entirely free and open as regards the possibility of doing something else for this patient other than amputation, because it is a great success and glory to surgery when we are able to save a limb like this; and it would have been a great matter in this case to have been able to assure the patient that there was a very reasonable chance of his getting well without amputation of the limb. I did not, however, feel that there was any likelihood or chance of that taking place; and I will tell you how I came to this conclusion.

The first thing about the case was the great length of time during which this femur had been affected. That is an element which students scarcely ever allow sufficiently for in a case of this kind. The femur had been broken thirty years before; and there is no question, considering that abscesses had formed at that time, that this was the origin of the mischief about the bone, perhaps aided by other causes. We had, then, a history of thirty years of more or less inflammation of the femur going on from some cause or other. What could that possible cause be? Of course there was the severity of the injury, upon which we must lay great stress, and the fact that the patient had rheumatic fever when a boy. Something, I think, must be attributed to a constitution in which there is a tendency to rheumatic fever. I think that such

patients are far more likely to undergo sclerosing inflammations than cases in which there is no rheumatic diathesis.

Some of you may say, "Yes, and he had a syphilis twenty-five years ago," but the syphilis was after the injury; and so I think, although we must regard the syphilis as another influencing cause, we must not lay any undue stress upon the fact.

The length of time during which the trouble had been going on was, I repeat, an important element to me in deciding what the nature of the lesion was. It seemed to me to be not a case of ordinary central necrosis, but a sclerosing inflammation of the lower end of the femur. I said in the theatre that there could be very little question that the whole of the lower end of the femur was in a condition of chronic osteitis, which generally means a sclerosing osteitis, the bone becoming denser in structure, so that parts naturally cancellous lose that character, and the bone throughout its substance takes on an ivory-like consistence. Think what it means to have a sclerosing osteitis going on in a bone, to the surface of which sinuses run down, and which is exposed to a large extent in a cavity containing foetid pus. In the first place, from the foetid pus we have a septic character given to the osteitis, and thus the process becomes, to a certain extent, allied to infective necrosis. Secondly, where sclerosing change is taking place, the blood vessels become encroached upon, and more or less obliterated, so that the Haversian canals instead of being of good size are very much diminished in calibre, and the supply of blood becomes in parts almost completely cut off. The nutrition then of considerable portions of the bone takes place by imbibition rather than by the circulation of blood through considerable channels. Hence there is a great tendency to necrosis in such bone from deprivation of proper blood supply. That is an important element indeed in the history of these cases. I should like you to associate that fact with the fact which I told you about the other day in my lectures on tumours of bone, because there are certain features about a case like this, which are interesting in relation to those hard ivory-like tumours found especially in connection with the frontal bone. I told you with reference to these ivory-like tumours of the frontal bone that one of the results which could be occasionally looked for was a necrotic change. Occasionally we find these ivory-like exostoses absolutely separating of themselves, sloughing away

from their base of attachment, the reason being that the gradual growth of this ivory-like structure encroaches upon the nutrient channels running into them, and obliterates them so that the bone dies, and the exostosis separates of itself. I told you that a mode of treatment which had been proposed for these tumours (which are often difficult to take away), was by making a number of drill apertures around the margin, partly through the dense bone and partly through the more healthy bone around it; and that the way in which this acts is that it disturbs the nutrition of the exostosis, and so facilitates the process of separation. These are two very interesting facts derived from the surgery of tumours of bone, which we can apply to a case such as this. I must also ask you to note that at a metropolitan hospital in the hands of an able surgeon, this patient was operated on for necrosis of the femur, an operation which practically, we may say, failed. The reason why it failed I must explain later on. In order to understand it fully, I think perhaps we had better go on to the details of the operation, and then to the appearances we found in the amputated limb.

I decided, then, that in this case it was necessary to remove the limb. The amputation was commenced so as to divide the bone just above where it was most thickened, that is to say, about six or seven inches above the knee joint. I made a long anterior flap in this case as the principal suppuration was towards the posterior aspect of the limb, and a comparatively short posterior flap so as to clear, as far as possible, any foul cavity which might exist in connection with the necrosis. When we got down to the bone I had to extend the flap a little higher in order to take in a little more bone, since we found the portion of bone we had reached was considerably thickened. Even then, on section of the bone, I found I had cut it across at a point where it was still undergoing the sclerosing change, so I removed three-quarters of an inch more of it. Afterwards the ordinary routine treatment was observed. We found it somewhat difficult to arrest the hæmorrhage; there was so much dense connective tissue (the product of former attacks of inflammation) running along the sheath of the femoral artery that the vessel was difficult to lay hold of and to separate from the surrounding structures. To overcome this difficulty, I found it was almost absolutely necessary to under-run it; this controlled the bleeding effectually. The flaps were then sutured, a drainage-tube introduced,

and the limb dressed with the ordinary antiseptic materials.

The registrar took the limb away in order to make a section of it for after-study. The next day I was told by the dresser that it was a beautiful case of central necrosis. It took my breath away a little, because that was not at all what I had expected or hoped to find. I said to myself, "Central necrosis! Then I have made a mistake; perhaps after all I ought to have done what the surgeon did elsewhere, and tried to take the dead bone away."* But it occurred to me that if this were an ordinary case of central necrosis why had the surgeon failed to cure the patient by his operation? We went to see the specimen. Here it is, on the table before us. It is one of the most interesting that you can possibly study. We found that although there was a necrosis in the centre of a cavity in the femur, the condition was not one to which the term "central necrosis" is usually applied. The process which gives rise to central necrosis is acute septic periostitis, or osteo-myelitis. It arises from one of those septic causes which are so very common, and which are related to some of the zymotic fevers (*e.g.*, measles).

In this way a periostitis develops itself, the pus formed in this process collects between the bone and periosteum, lifting the latter membrane off the bone, and thus destroying its vascular connections. Hence necrosis of this portion of the bone results. In the course of three or four months after the commencement of the inflammation, ossification takes place in the deeper layers of the periosteum, and a sheath of new bone is formed. This sheath encloses a cavity containing pus, in the interior of which is the central sequestrum. That is central necrosis due to acute septic periostitis. You may ask, Why is not this such a case? Do not these necrotic portions of bone result from acute septic periostitis? No, they do not—at least not mainly. Examination of the specimen shows that there is another cause for the necrosis. Central necrosis is usually an acute change. The periosteum gets very quickly lifted off the bone, which is left very much in the condition in which it was at the time

* The term is no doubt somewhat loosely applied in surgery. Sometimes it means *any* loose piece of necrosed bone, surrounded by a sheath of new bone. Sometimes it is restricted to a necrosis resulting from a localized osteo-myelitis of the cancellous tissue. More often it is applied to the central piece of necrosed shaft of a long bone, surrounded by new bone developed from periosteum—the whole resulting from acute infective periostitis. It was this form that I understood the dresser to refer to.

of the supervention of the inflammation. I show you here a specimen from the museum, in which the shaft of the bone is left precisely as it was at the time the inflammation commenced. The necrosed portion is here placed in the centre of young living bone of very vascular cancellous structure. Look at the difference between the two specimens. In the specimen from the patient I am lecturing on, a portion of the sequestrum has been cut in longitudinal section by the saw. It is still firmly in connection with the living bone at one place. Instead of being more or less cancellous in its structure, it is dense and hard like ivory. That is the crux of the whole condition. How did it get dense and hard like ivory? Because, of course, at one time or another this bone was slowly undergoing a process of inflammation, by which the Haversian canals were becoming gradually diminished in size, fresh osseous material was being continually deposited about it, until it had undergone this sclerotic change.

I must now explain to you why the previous operation for the removal of the sequestrum failed. If you examine the specimen again, you will find that a quantity of the *living* external bone is in almost precisely the same ivory-like condition as the necrotic portion internally. I want you to connect these two facts together, and also to the operation done six or seven years ago, when he was supposed to have a central necrosis requiring removal. You will see very quickly what took place. Just suppose, for an instant, I determined to repeat that operation, to cut down upon the bone and remove the necrosis. What would have been the almost inevitable result of that operation?

I do not ask you to consider now the risks of cutting into a cavity like this containing the most septic pus imaginable, risks of pyæmia and septicæmia, which would be very great indeed in a case of this kind. But supposing I tried to lay open this cavity, I should have, in the first place, to lay open the sinuses to a very great extent, making probably an incision on the outer side of the thigh to get at the bone more satisfactorily. I should then have had to trephine or cut through this very dense ivory-like bone, in order to lay bare the cavity, and as this cavity is of very irregular size (there are two in the specimen before us) I should have had to cut through this ivory-like bone very freely indeed. Supposing I had laid bare the cavity quite satisfactorily, and taken away all the dead internal bone, leaving the ivory-

like external living bone, what would have been the result? Just as we find drilling causes necrosis and favours exfoliation, just as the natural production of this ivory-like exostosis tends to obliteration of the vessels and sloughing, so the mere fact of cutting through the ivory-like bone would have determined the death of the portion of bone cut through. The skin wound would have united, and we should have had the repetition of the whole process again; in the course of the next year or two the patient would have had new sinuses form, in which bare, dead bone would have been found; a necrosis brought about by the surgeon's operation, mark you. In such ivory bone there is not sufficient vascular supply to permit the formation of healthy granulation tissue. A wound in such bone cannot heal up. A fresh mass dies, and the necrosis repeats itself. Such I believe was the very process which took place after the operation six years ago. Probably the surgeon at that operation took away all the bone necrosed at that time, but in so doing he damaged the nutrition of the bone he cut through to reach the necrosis, and hence ensued a further necrosis.

When you have bone of such density, with a nutrition so feeble, the only thing that can be done in a case like this is amputation of the limb. The case, as far as the limb is concerned, is hopeless; and to attempt to save such a limb is only exposing the patient to risks not only of septicæmia and pyæmia, but others, after these more acute risks have passed away. Remember we have here a patient suffering from a most highly septic discharge probably for four or five years. A patient otherwise healthy is not improved by such suppuration; and this patient who has had syphilis, which may have had some effect in the production of the bone disease, may possibly become the victim of lardaceous disease of the liver, kidney, and intestine, so that his strength may be gradually drained away, and he himself be killed in that way.

I think, therefore, that I have shown you reasons quite sufficient for saying decidedly that the only treatment here which was at all likely to be successful was amputation of the limb. And I am glad to tell you that he has been going on very well since the operation. We had a little trouble during the operation with the very numerous blood-vessels which had become enlarged during all this process of suppuration, so that a larger number of ligatures were required than usual, but there has been no

hæmorrhage or other bad symptom since the operation, and I think we may hope for the best.

It is always well to look forward a little, and I ought perhaps to temper this little vein of congratulation by just a little croaking over the future. Is it absolutely certain that this amputation will heal up entirely and the patient get a perfectly good stump? I should be inclined if this had been done in private practice to throw in a word of caution and warning to the patient and his friends. Although we cut through the bone as far above the sclerosing portion as possible there was still a considerable amount of sclerosing bone on the femur in the stump. Hence it is possible that a portion of this sclerosing bone may die from my operation, and may form a small tubular sequestrum in the stump. Even if this takes place it will probably be easily removed now that the amputation has been done. But it is necessary to recollect that such a result is possible.

Mr. Rake, our surgical registrar, reports the condition as follows: "On making a longitudinal section through the portion of the femur removed, three inches from the articular surface a triangular cavity is found lined with velvety pyogenic membrane, and measuring two inches in length and one inch and a half from before backwards. This cavity was found to contain a large fixed, greenish-grey sequestrum" (the very words suggest a suppuration of the most abominable odour) "besides smaller fixed and loose ones, and also some extremely foul-smelling pus." (The point about that is, remember, that my dresser tried carefully to wash this out, and as far as possible to disinfect the cavity; and yet the report says that after the operation,—so difficult is it to disinfect these cases,—there was this foul-smelling pus in the interior.)

"On dissecting out the sinuses on the back of the leg, the more anterior and internal one was found to communicate with a cloaca on the lateral surface of the femur, while the more posterior communicated by a circuitous course with a cloaca on the posterior surface.

"The triangular cavity above mentioned communicated above with the cancelli at the lower end of the medullary cavity of the femur." (I do not entirely agree with this. These cancelli towards the sequestrum cavity are distinctly much more indurated than the cancelli in the epiphysis, for instance, or those at a greater distance from the cavity. I do not think we can say they communicate with the cancelli of the medullary cavity,

for if they did we should have had a greater amount of sapræmia than we had in this case) "although there was no suppuration in the medullary cavity above the sequestra-containing cavity."

(So that it is perfectly clear the medullary cavity *was* cut off from this foul-smelling cavity.)

"Posteriorly the cavity was shut off from the popliteal space only by a tough fibrous membrane" (which I should conclude to be either the periosteum or some thickening of the fascial tissue or the ligaments. On introducing the probe I felt no bone on the posterior aspect, and the impression left on my mind was that there were some fascial or periosteal tissues of the popliteal space which the probe directly abutted against posteriorly. That is very fairly borne out by the description here—"shut off from the popliteal space only by a tough fibrous membrane.")

"Anteriorly and postero-superiorly it was bounded by extremely dense ivory-like bone. The cancellous tissue below the cavity seems to have been rarified; one large space filled with fat measuring $\frac{3}{4}$ in. in diameter."

"There is an especially large amount of compact bone running along the posterior surface of the bone owing probably to the patient walking with his knee bent."

(When we examined the patient, the limb was not absolutely straight but slightly bent at the knee. It seemed to have gone backwards a little from its correct position, as regards the femur, just as if there was a slight condition of backward displacement. There was, however, no true backward displacement; the apparent slight backward displacement being no doubt brought about by a slight curve in the lower end of the femur at the point where it had undergone all the inflammatory changes.)

"The second portion of bone removed measured $\frac{3}{4}$ in. in length and shows compact sclerosed tissue."

There was one special risk in this operation which I should like to caution you about, namely, that wherever you get a very foul cavity in communication with necrosing bone, you should be a little guarded as regards the nature of the amputation you perform. I chose the long anterior and the short posterior flap, hoping to clear these cavities, and fortunately we did clear them. I do not think, therefore, we are likely to get any bad effect from these cavities in this case. But it might have been that these cavities ran up higher on the posterior aspect of the femur than I had anticipated,

and in making the short posterior flap I might have gone across one of them. If I had done so, it would have immeasurably increased the risks to the patient. The cavity contained foul pus, which would have infected the whole surface of the freshly-cut muscle, fat, fascial vessel, and nerve tissues, which form the flaps. However carefully you tried to asepticize this discharge you would fail practically in destroying all the septic material. The chances are that the wound would have become septic, and we should have had (for your advantage in study, no doubt, but to the great disadvantage of the patient) one of those cases of septic amputation such as we saw years ago before Sir Joseph Lister introduced antiseptic treatment. That is no imaginary picture. Some three or four years ago I had to amputate low down the lower third of the thigh in a case in which there was old hip-joint disease,—one of the cases which last year I brought before the Medico-Chirurgical Society as showing the effects of an amputation of the limb below the seat of the disease in influencing the disease itself. In doing so, quite ignorantly on my part, I cut across a suppurating of great extent, the sinus extending low down from a necrotic portion of the os innominatum high up, and discharging foetid pus. We attempted to wash it out and asepticize it thoroughly. Unfortunately we were not successful. Consequently primary union did not take place, and the patient's temperature rose to $103-4^{\circ}$; and we had evidently a case of septic osteo-myelitis. One of the not infrequent results, such as we saw them in the old times followed upon that. Some days after the amputation, secondary hæmorrhage occurred,—no doubt from the vessels undergoing septic changes, softening, and not closing and healing as they do in an aseptic stump. Recognizing what the cause was, we separated the whole of the flaps, secured the vessel again, and without attempting any more primary union, we plugged the flaps with antiseptic material so as to make them granulate from the bottom, and washed out the whole interior with an antiseptic fluid whenever the wound was dressed.

That had the effect of preventing any more absorption, and by applying the antiseptic fluids to the whole surface of the stump and to the sinus itself we were successful in getting the whole thing to close up. In that way the case was saved. But you can easily understand how great would be the risk in a case of this kind. We might have saved

this patient's life in the same manner, but if the stump had become septic the risks would have been immeasurably increased. Therefore, if you have to amputate in one of these cases of necrosis, try to avoid offensive pus and sinuses. If you cannot avoid them, be prepared for the necessity of opening up your stump, and asepticizing it as described.

ON PELVIC ABSCESS.

An Address delivered at Birmingham at the opening Meeting of the Midland Medical Society, Nov. 2, 1893,

BY

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I CONSIDER it a great distinction to have been invited to deliver this inaugural address. When I looked down the list of those who have had a similar honour paid to them in previous years, and remembered how almost every one of them had made it an occasion for adding something of sterling and permanent value to the literature of our profession, I realized (with much misgiving as to my competence for the task) how serious a duty I had undertaken.

In the address in obstetrics and gynaecology which I had the honour of delivering at the recent Meeting of the British Medical Association at Newcastle, I spoke of the debt that gynaecology owed to the enterprise of the modern abdominal surgeon, in regard more particularly to the light that had thereby been thrown on the pathology, diagnosis and treatment of inflammation of the Fallopian tubes. Remembering that my address this evening would be delivered in Birmingham, the city where, for many years, abdominal surgery received its chief impetus, it seemed to me natural and fitting that I should utilize the occasion for calling attention to some of the many *other* directions in which our knowledge of pelvic disease has recently been much advanced, and in which the advance has been due, if not entirely, certainly very largely, to the teachings of abdominal surgery. The subject I have chosen is pelvic abscess, or, to speak more correctly, pelvic suppuration, in the female.

Until quite recently, all forms of pelvic suppurations were styled, indiscriminately, pelvic

abscess, and our ideas as to their etiology and pathology were exceedingly vague. It was generally believed that there were two forms of pelvic abscess, the one and more common form having its seat in the pelvic connective tissue, the other and rarer form being intra-peritoneal, and being located for the most part in Douglas's pouch, where the pus, having in the first instance accumulated in that pouch by the law of gravitation, eventually became encysted by adhesions. The one was held to be the result of cellulitis, the other of peritonitis, both of which were regarded as idiopathic affections, capable of being induced either by exposure to cold (especially during menstruation and after parturition and abortion), or by direct injury received either during labour or in the course of surgical manipulations, conducted through the vagina. The changes that have been brought about in our views respecting suppuration in general, and pelvic inflammations in particular have of necessity affected our notions as to pelvic abscess. We now know that suppuration in the pelvis as in other parts of the body is always an infective process, and can never have either a purely catarrhal or a purely traumatic origin. Neither cellulitis nor peritonitis can any longer be regarded as an idiopathic affection. Catarrh and injury may be, and often are, factors in their production, but they are not the essential factors. There must always be an infection of some kind. Our knowledge, too, of the *morbid anatomy* of pelvic abscess has received important additions. We now know that the immediate sources of pelvic suppuration are much more numerous than they were supposed to be, and that, although it may be convenient to retain the term pelvic abscess as a specific name for cases of suppuration in the connective tissue of the pelvis, and as a provisional diagnosis in other cases of pelvic suppuration, where the precise locality of the lesion has not yet been discovered, the time has come to discard it as a general term, and to aim, in every case of pelvic suppuration, at a more exact diagnosis than the use of such a term would imply. I am no advocate for pretending to a refinement of diagnosis that is unattainable, but when I see a hospital physician never venturing further in the diagnosis of a skin disease than to call it an "eruption," and another entering every inflammatory affection of the respiratory tract in childhood under the safe heading of "cough," and a third publicly advocating the application of the old term pelvic abscess to

every form of pelvic suppuration; I cannot but feel that in this matter of diagnosis modesty and caution may be carried too far.

ABSCESS IN THE CONNECTIVE TISSUE OF THE PELVIS.

The simplest form of pelvic suppuration is that which occurs in the connective tissue, and it is this form to which I should like to see the use of the term pelvic abscess restricted. It will immensely facilitate our study of this affection if we recognize, once for all, that inflammation of the connective tissue in the pelvis differs in no way from inflammation of the same tissue in other parts of the body, which are more accessible to sight and touch. I was glad to see the attention of gynecologists very forcibly called to this point by Prof. Wm. Keiller of the University of Texas, in the September number of the *Amer. Journal of Obstetrics*. Primary cellulitis, as we know it in other parts of the body, is always an acute affection, and is always septic in its origin. Prof. Keiller takes as an example the ordinary clinical course of a septic wound of the palm of the hand. The process in pelvic cellulitis is precisely analogous. "The cellular area of the pelvis is shut off from the perineum by the pelvic fascia. This strong aponeurosis is attached to the pelvic wall between the pubic bones and bodies of the ischia along that thickening of the obturator fascia known as the white line. From this it passes as a continuous sheet over the levator ani and coccygeus to the vagina in front, and the rectum and coccyx behind. Behind the pubic symphysis it is closely blended with the vaginal orifice under the name of the triangular ligament. All inflammatory exudation connected with the female genitals above the vulva takes place above this strong fascia; and as, in a septic wound of the palm of the hand, the exuded serum or lymph is forced to find accommodation on the back of the hand by the strong palmar fascia, and the way in which the skin is bound to it, so the pelvic fascia forces the exuded fluids in septic wounds of the deeper genitals, to find accommodation in the loose tissue above it. . . ." This cellular area has for its upper boundary the peritoneum, which can be readily stripped from the whole of the pelvis except in the following situations, viz., the anterior surface and sides of the rectum, the upper part of the posterior wall of the vagina, the posterior surface and fundus of the uterus, the anterior surface of

the body of the uterus, and the posterior surface of the bladder. As all these parts, where the peritoneum is firmly attached, are situated more or less in the middle line, it follows, that the cellular area of one side scarcely communicates with that of the other, except along the tract which lies between the upper part of the cervix and the bladder, so that cellulitic exudations are mostly limited to one side. The most common source of such exudations is septic absorption through lacerations of the cervix, and of the upper part of the vagina, occurring during labour, the latter accident accompanying the use of the forceps much more frequently than is supposed. I have over and over again, in cases of pelvic cellulitis seen in consultation, found wounds of the vagina that had been entirely unsuspected by the medical practitioner in attendance, and that had evidently been caused by the projecting edge of one of the blades of the forceps. Such wounds, if they remain aseptic, rapidly heal; but it often happens that septic matter finds its way into them, and then pelvic cellulitis results. Only less important than parturition in respect to the etiology of this affection, are the various surgical measures practised on the vagina and cervix. Before the necessity of rigid aseptic precautions was understood and generally acted upon, the most trifling surgical proceedings in these parts were apt to be followed by an attack of cellulitis. With regard to abortion, I am inclined to believe that septic infection following abortion seldom, if ever, takes the form of pelvic cellulitis, for the simple reason that, there being no over-distension of the cervix, or wounding of the vagina by the use of instruments, injuries to those parts are less likely to occur. This impression is abundantly borne out by my own experience. In examining the records of St. Thomas's Hospital for the past five years, I find that during this period there have been twenty-two unequivocal and uncomplicated cases of cellulitic abscess in the pelvis. Of these not a single one followed abortion. Twenty-one were the result of parturition, and the twenty-second, the cause of which was not discovered, occurred in the fifth month of pregnancy. In connection with this subject of abscess in the pelvic connective tissue, Prof. Keiller very properly calls attention to the rôle played by the lymphatic vessels and glands. He points out that nothing is more common, as the result of a neglected septic wound of foot or hand, than to have a suppurating gland in the

groin or axilla, and asks if it is "to be expected that the pelvic glands will always escape in vaginal or cervical wounds." I feel as certain as he does that they do not escape, and I can call to mind several cases where, from the position of the abscess, I have no doubt that there was suppuration of the hypogastric glands, those, namely, which surround the iliac vessels at the sides of the pelvis, and receive the lymphatics from the cervix and the upper three-fourths of the vagina. "Of course," as Prof. Keiller says, "we cannot palpate a large and tender gland" in such a situation, "and for this reason we are very apt to forget its presence."

Where do cellulitic abscesses in the pelvis usually point? In the great majority of cases the spread of the inflammation is in a forward direction, from the base of the broad ligament, where it starts, to the connective tissue lying beneath the peritoneum which forms the floor of the anterior pelvic fossa, stripping up the peritoneum where it becomes reflected on to the anterior abdominal wall and forming a broad strip of induration along and parallel with the upper border of Poupart's ligament. When suppuration occurs, the skin over the induration gradually becomes oedematous and pointing usually occurs at a spot almost immediately above the centre of Poupart's ligament. It will be seen, from the accompanying table, that, of the 22 cases of un-

TABLE I.

Cellulitic Abscess in Pelvis, 1889-93.

Pointed above Poupart's ligament—			
Right side	9
Left side	9
		—	18
Pointed over iliac crest	1
Opened before pointing occurred; abscess			
situated on posterior wall of pelvis	3
		—	22

complicated cellulitic abscess treated during the last five years in the women's ward at St. Thomas's Hospital, the abscess pointed above Poupart's ligament in 18, nine times on the right side and nine times on the left. This mode of extension of the inflammation is often described as "following the course of the round ligament," but I know of no evidence to show that the round ligament plays any part in determining the course of the inflammation. The study of frozen sections has taught us that, at the end of pregnancy, the broad ligaments have been drawn upwards by the growing uterus, so that the base of the ligament

is at the brim of the pelvis and the peritoneum no longer descends laterally into the pelvis at all. The whole of the lateral space below the pelvic brim is, at this period, occupied by cellular tissue, the quantity of which has become enormously increased (see "Barbour's Sectional Anatomy of Labour," pp. 16 to 18). There is, therefore, no need to invoke the intervention of the round ligaments to account for the inflammation passing forwards from the base of the broad ligament to the tissues just above Poupart's ligament. These parts already lie in close juxtaposition, and the extension of inflammation from one to the other really needs no other explanation.

In exceptional cases the inflammation passes round to the loose tissue in front of the bladder, and the abscess points above the symphysis, or, mounting up between the anterior parietal peritoneum and the sheath of the rectus, points at the umbilicus. In three of the 22 cases tabulated, the abscess formed at the back, instead of at the front, of the pelvis, probably owing to implication of the hypogastric glands, and was opened by making an incision above the anterior superior iliac spine and dissecting inwards, beneath the peritoneum, to the pelvic brim. It is probably in cases of this kind that the suppuration occasionally extends upwards along the sub-peritoneal tissue of the iliac fossa and even into that of the loin, pointing either at the iliac crest, as in one of the cases in the table, or above it. The text-books speak of the pus, in such cases, following the course of the psoas muscle; but when matter burrows along the psoas it is not from a cellulitic abscess, but from dead bone. It is along the blood-vessels and other parts, such as the ureter, that actually lie in the connective tissue of the pelvis, and are accompanied by a prolongation of it as they enter and leave the pelvis, that the pus finds its way.* This is not only true of the cases in which suppuration extends to the iliac fossa and the loin, but also of those where the pus leaves the pelvis by the sciatic notch, or, passing beneath Poupart's ligament, points in Scarpa's triangle. In the former case, it follows the course, not of the obturator tendon, but of the sciatic and gluteal vessels, and, in the latter, it is the femoral vessels, and not nerve or tendon,

as is sometimes stated, that direct the course of the abscess.

It is commonly stated that cellulitic abscesses frequently burst into the rectum, the vagina, and the bladder. This statement appears to me to rest on very slender foundation. Many of the cases quoted in its support belong to an era when little was known of the pathology of pelvic inflammation, and, on carefully reading them in the light of our present knowledge, it is easy to see that at least a considerable number reported as cellulitic abscesses were really cases of intra-peritoneal suppuration originating in suppurative disease either of the tubes or ovaries. This is notably true of a case of my own, published twenty-two years ago, which, though I have now no doubt whatever as to its having been a case of intra-peritoneal suppuration from acute tubal disease, was described, in accordance with the crude gynecological pathology of the time, under the head of pelvic cellulitis, and takes its place in Delbet's valuable Monograph as a well-authenticated instance of the spontaneous opening of a cellulitic abscess into the rectum. At the same time it is only fair to say that there does not appear to be any anatomical reason why cellulitic abscess should not *occasionally* discharge themselves into the rectum, the vagina and the bladder, and that some of the cases in Delbet's collection appear to be genuine examples of such an occurrence.

The next table shows, in regard to the twenty-one cases in which cellulitic abscess followed parturition, the period that elapsed between delivery and the pointing or opening of the abscess. It will be seen that, judging from the

TABLE II.

Period after delivery when abscess pointed or was opened.

Over 5 and under 6 weeks	= 1
" 6 " 7 "	— 4
" 7 " 8 "	— 2
" 8 " 9 "	— 3
" 9 " 10 "	— 5
" 10 " 11 "	— 2
" 11 " 12 "	— 3
" 12 " 13 "	— 1
" 14 " 15 "	— 1
Total ...	21

small number of cases here recorded, the usual time for the abscess to point is between the seventh and the twelfth week. The earliest period at which pointing occurred was five weeks, the latest fourteen.

The treatment of cellulitic abscess in the pelvis,

* See Anderson (W.) and Makins (G. H.), The Planes of Sub-peritoneal and Sub-pleural Connective Tissue, with their Extensions. "Journal of Anatomy and Physiology. Vol. xxv., Pt. I., Oct. 1890, p. 78.

i.e., of true pelvic abscess, is summed up in two words, incision and drainage. Abdominal section is here entirely uncalled for. The abscess should be opened as soon as ever fluctuation is detected or there is the faintest indication of pointing. In ordinary cases the drainage-tube is only required a very few days. Unlike other forms of suppuration in the pelvis, cellulitic abscess, in my experience, tends, when once the matter has been set free, to complete and rapid recovery. I have never seen troublesome sinuses form, and the tendency to lateral displacement of the uterus from subsequent contraction of cicatricial tissue has, I believe, been much exaggerated. This affection offers no bar to subsequent conception, and when pregnancy again takes place its normal course is not interfered with. All this is in strong contrast to what occurs after other forms of pelvic suppuration.

I have had no experience of cellulitic abscesses pointing in the vagina, and therefore have never had occasion to open one there. Frequently I have found, in cases of pelvic inflammation, soft fluctuating swellings depressing the vaginal vault, both laterally and posteriorly, but such swellings have invariably proved to be other than cellulitic, and I have often had cause to be thankful that I had not been tempted to open them from below.

When I said just now that abdominal section was never required in cases of cellulitic abscess, I had not forgotten Mr. Lawson Tait's historical paper, read before the Royal Medical and Chirurgical Society in 1880. The cases of pelvic abscess, however, that were described in that paper were not cellulitic, but were examples, apparently, of suppurating hæmatoma of the broad ligament, and, as such, do not come within the category of which I have been speaking. They belong rather to the group with which I come now to deal, and of which I agree with Mr. Tait in believing abdominal section to be the only proper treatment.

Other forms of Pelvic Suppuration.

Passing now to other forms of pelvic suppuration, it is desirable in the first instance to obtain some idea of their relative frequency. With that object I have tabulated eighty-three cases in which I have performed abdominal section, and found suppurative disease within the pelvis. The number is not large, but it is sufficient to give some indication of the main sources of intra-pelvic suppuration, and

of their relative frequency. The eighty-three cases may be classified as follows:—

TABLE III.

Purulent salpingitis (including pyosalpinx)	37
Purulent salpingitis, with suppurating cyst of ovary (eight communicating) ...	13
Suppurating cyst of ovary ...	17
Tubercular disease of tube ...	3
of ovary ...	3
of tube and ovary	1
	— 7
Disease of vermiform appendix ...	1
Suppurating retro-peritoneal cyst ...	1
Suppurating lumbar gland ...	1
Undetermined ...	6
	— 83

From this table it is abundantly clear that purulent salpingitis is much the most frequent source of intra-pelvic suppuration. It was met with in upwards of 60 per cent. of the cases. In 44½ per cent. no other suppurative disease existed, and in the remaining 15½ per cent., although associated with suppurating cyst of the ovary, there was strong evidence of the suppurative change in the ovary being secondary to the purulent inflammation of the tube.

Having so recently spoken of the pathological importance of the Fallopian tubes in connection with pelvic peritonitis, I need not, on this occasion, further insist upon it. I may, however, point out that the reason why there is no separate mention, in the table, of cases in which encysted intra-peritoneal collections of pus were found, is that such collections are almost invariably a mere complication of purulent salpingitis. We are in the habit of thinking of suppurating Fallopian tubes as closed sacs of pus, the pus being pent up in the tubes by occlusion of the abdominal ostium. But it must be remembered that in the earlier stages of purulent salpingitis the fimbriated ends are open, and the pus is free to discharge itself into the peritoneal cavity. In a paper recently read before the Obstetrical Society of London, I described a case of gonorrhœal salpingitis, in which the fimbriated ends of both tubes were open, and were discharging their purulent contents into Douglas's pouch, which had become closed in by adhesions. The pouch formed a little reservoir of pus, fed by two supply pipes, and with no outlet. Had the case been allowed to go on, no doubt ulceration into the rectum would have taken place. It was particularly interesting to see one of these intra-peritoneal collections of pus in actual process of formation.

(To be concluded).

THERAPEUTICAL NOTES.

Nitro-Glycerine Hypodermically in Epilepsy.—Although Nitro-glycerine has been used to abort the attack in epilepsy, Dr. H. Elliott Bates states that it has not been employed after the attack has been established and when the sufferer lies where he has fallen with rigid limbs and unconscious of all surroundings. He has used it in the above stage, and cites some cases in support. In the first case the patient had been suffering for four years, and was seen under the effects of a fully developed epileptic seizure. A hypodermic injection of 1-100 grain of Nitro-glycerine was given, and before the needle could be withdrawn, total relaxation took place, consciousness returned, and the patient called for a drink of water. In the second case, also, the administration of the drug was followed by speedy and very satisfactory results. In a third case the method proved of great value, as the patient, while in the attack, was very violent, and an object of terror to his family and the neighbourhood. The use of the drug was followed by a prompt return of consciousness. The method has been tried in nine other cases, also without a failure. Once only a second dose was required. In all the cases the after-effects of the attack were markedly lessened, the patients recovered without the fatigue and general demoralization, and the sudden transition from an object of terror to a rational being has been of considerable value to patient and physician. It is *not* claimed for the method that it is curative. It shortens the attack, saves fatigue, and has some influence upon the frequency of attacks. The after-treatment consists of the Bromides in a bitter infusion, and the use of minute doses of Nitro-glycerine.—(*N. Y. Med. Jour.*)

The Treatment of Tic-Douloureux.—Dr. Jarre read a paper before the Académie de Médecine de Paris on September 5th, on the subject of tic-douloureux, in which he attempted to establish on a definite basis not only the pathology and mechanism of production of this painful affection, but also the treatment, and he detailed the lines which he had adopted. He maintained that cicatricial lesions of the nerve were the cause of a large majority of the cases, and that the treatment consisted in attempting to remove the cicatrices. As a rule these lesions were situated

in the alveolar region at the terminal extremities of the nerves. The most common causes of the cicatrices were chronic alveolar-dental inflammation and conditions brought about by the faulty development of the lower wisdom teeth. The treatment indicated in such cases is to remove a portion of the alveolus, together with the cicatrix which it encloses. The operation is best performed in three stages, firstly, the mucous membrane and the periosteum covering the portion of the alveolus which is to be resected are turned aside; secondly, the piece of bone is removed; thirdly, the surface of bone exposed is well scraped. The wound is then washed out and dressed with a pad of cotton-wool soaked in an antiseptic solution. The immediate results of the operation in Dr. Jarre's patients were at first a diminution in the number of attacks of pain, and finally, in four or five days, a total cessation of the symptoms. Ten cases, in which all other methods of treatment had been previously tried without success, completely recovered after this operation.—(*Med. Record.*)

Iodized Collodion for Ringworm.—Butte strongly recommends the following for the treatment of ringworm:—Dissolve 10 grains of Iodine in 3 drachms of rectified spirit, add Collodion an ounce and a half, Venice Turpentine 24 grains, and Castor-oil half a drachm. Apply to the patch for three or four successive days till a thick and adherent layer is formed. Remove in fifteen days, and wash in 1-500 solution of Mercuric Chloride. (*An. de Dermat. de Syph.—Chem. and Drug.*)

FORMULÆ.

Chronic Tonsillitis:—

R	Acid. Tannic	gr.xv
	Tinct. Iodi	gtt.ij
	Aquæ	℥vj
	Glycerini	℥ss

M. Sig.: Tablespoonful every three hours.
(*Med. Rec.*)

Neuralgia:—

R	Acentalid	gr.j
	Quinin. Sulph.	gr.j
	Cocain.	gr. 1/10

For one pill. Give every hour.
(*Med. Rec.*)

REVIEWS.

On Diseases of the Lungs and Pleuræ, including Consumption. By R. DOUGLAS POWELL, M.D. Lond. (Fourth Edition. H. K. Lewis. 1893.)

Published at 18s.

This work gives a comprehensive account of the diseases of the lungs and pleuræ, including the essential points in the anatomy and functions of the organs, and the physical examination of the chest.

Though sufficiently full to be a most valuable book for specialists in lung diseases—for whom it is particularly suited—it will be read with advantage by all medical men.

The first chapter deals with the anatomy and physiology of the lungs. This careful summary of the structure and functions of these organs forms a necessary preliminary to the understanding of the effects of disease. The second chapter, on the physical examination of the chest by an expert in such examination, who is also an experienced teacher, cannot but be valuable. The discussion on the *rationale* of the sounds produced in the chest in health and disease is very complete.

Microscopical examination of the sputa receives adequate notice in a special chapter. The account of diseases of the pleuræ is followed by an extremely practical chapter on the surgical treatment of pleuritic effusions.

Bronchitis, emphysema, pneumonia, and asthma are fully discussed, and a chapter is devoted to the surgical treatment of pulmonary cavities. Hydatids of the lungs and actinomycosis also receive full notice.

Phthisis occupies twenty chapters; and in these 230 pages will be found a most complete account of the etiology, pathology, and symptomology of this disease, with the treatment both prophylactic and curative.

The illustrative cases add to the interest of the description of the various forms of the disease. The whole work is worthy of the high reputation of the author as an authority on diseases of the lungs. That it has reached the fourth edition shows that its value has been appreciated; and as this edition has been brought up to the present state of medical knowledge, the book keeps its place as a standard work on the subject with which it deals in so masterly a manner. The illustrations are in keeping with the rest of the work, and a good index adds to its value as a book of reference.

Anæsthetics and their Administration. By FREDERICK W. HEWITT, M.A., M.D. Cantab. (Charles Griffin & Co., London),

Published at 10s.

This book, the result of ten years practical experience by its author, is a valuable addition to the literature of the subject, being a most admirably arranged and exhaustive account of all questions involved. Dr. Hewitt has divided the book into four parts, each part being sub-divided into several chapters, and each chapter being arranged in suitable and comprehensive sections. Part I. is devoted to preliminary considerations. These are of a thoroughly practical nature, commencing with a short description of

the various anæsthetics proceeding from that to what may be termed the personal equation of the patient as regards his age, temperament, habits, and the points to be considered as to the influence of any organic disease with which he may be affected. Dr. Hewitt then puts before us the points to be noted as to the particular operation for which the anæsthetic is needed, not only as regards the period for which the anæsthesia is to be kept up, but also as regards the position of the patient during the operation, and further as regards the convenience of the operator. From here he passes on to the various considerations that must be taken into account in preparing the patient for the anæsthetic, as regards food, state of the bowels, and bladder. This part is brought to a close by a description of the various appliances and remedies which should be kept in readiness.

Part II. is devoted to the administration of the selected anæsthetic, and occupies the greater part of the book. The author describes fully the apparatus and methods of administration of each of the generally used anæsthetics. In each case he describes the effects produced, and other important details, such as the dangers and the after effects of each. Illustrative cases are added to each section, and add considerably to the value and interest of this very important part.

Part III. contains a description of the management and treatment of the difficulties, accidents, and dangers of general anæsthesia. They are arranged under the heading of minor difficulties, such as vomiting, retching, etc., those associated with respiration, and those associated with circulation. Here again Dr. Hewitt is very explicit, going thoroughly into the causation and treatment, and quoting illustrative cases.

Part IV. describes the condition of the patient after the administration; and contains practical advice as to the management of patients then.

It will be seen from our brief description of the leading features of the book that it is in every way a practical book of the highest use. It takes one, step by step, from the choice of the anæsthetic to the proper method of administering it; then it warns as to the dangers which may arise, and gives advice as to how these are to be dealt with, and finally gives explicit directions as to the after management. Throughout the book most careful attention to minute details is shown. It is well printed, and illustrated, and the index is well compiled. Its systematic arrangement, the care for details, and its lucidity render it a book which can be recommended as one of great use to all practitioners and students of medicine. The advance of all sciences connected with medicine and surgery is well illustrated by the teaching of anæsthetic administration at our schools, and those who wish to become acquainted with the scientific grounds for the use of suitable anæsthetics for different surgical procedures which have taken the place of the haphazard method once in vogue, cannot do better than make themselves thoroughly acquainted with the contents of Dr. Hewitt's admirable book.

DR. GOWERS will resume his weekly Clinical Lectures at the National Hospital for the Paralysed and Epileptic to-day, *Wednesday (November 8th) at 3 p.m.*

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 15, 1893.

ON PELVIC ABSCESS.

An Address delivered at Birmingham at the opening Meeting of the Midland Medical Society, Nov. 2, 1893.

BY

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(Concluded from p. 30.)

Suppurating Cysts of the Ovary, a form of pelvic suppuration frequently unrecognized.

Next to purulent salpingitis the most common form of pelvic suppuration, is suppurating cyst of the ovary. The frequency with which acute pelvic peritonitis is due to the presence of small suppurating ovarian cysts which, owing to their size, had not previously been known to exist, is, I believe, not generally recognized. At any rate, it has been quite in the nature of a revelation to me, as case after case has occurred in the course of my own work. I published in the Hospital Mirror of the "Lancet," of July 2 and 9, 1892, a series of six such cases, all of which happened to be under my care at one time. In two of them the disease was bilateral and the cysts of considerable size, yet not even in these had any abdominal swelling been observed previous to the severe attack of pelvic peritonitis for which the patients sought admission to the hospital. When peritonitis occurs in a patient with an ovarian cyst, large enough to attract attention as a distinct abdominal swelling, it is easy enough to recognize that the peritonitis is due to an inflammation of the cyst; but when such an occurrence takes place in a patient not known to have ovarian disease the source of the inflammation is almost certain to remain unsuspected, and the swelling subsequently observed is almost equally certain to be regarded as purely inflammatory, and unconnected with pre-existing disease. These cases are invariably sent into the hospital as cases of acute pelvic cellulitis. I feel sure that even when the abdomen has been opened, and the suppuration discovered, the true nature of the case has not always been made out. Many instances in which a collection of pus has been opened, and the cavity drained, and in which the

operator has been unable to satisfy himself as to the precise seat of the suppuration, have, I feel little doubt, been cases of suppurating ovarian cyst. The matting together of adjacent parts is of itself sufficient to introduce an element of confusion, and to render recognition difficult. But there is one condition that contributes to increase this difficulty more than any other, and that is the tendency of an ovarian cyst, when it becomes inflamed while still small enough to lie in the pelvis, to contract adhesions to the broad ligament, and, in the course of its enlargement, to draw the stretched and thickened broad ligament over it, until its anterior surface is completely concealed by it, as by a hood. Until an operator becomes familiar with this phenomenon, the condition which presents itself to his eye and touch, is exceedingly puzzling and misleading. What often happens is that, deep-seated fluctuation being detected, a trocar is passed *through the broad ligament* into the suppurating cyst behind it, and the cyst is emptied and drained under the impression that it is a collection of pus either in the broad ligament or behind the parietal peritoneum. Operators who have had frequent occasion to open the abdomen in cases of obscure pelvic suppuration will, I feel sure, recognize the truth of this description. The proper treatment of such cases, is, not to tap the cyst, but, having obtained access to it by the careful and patient separation of adhesions, to enucleate it, if possible entire, until there remain only the normal attachments, which can then be treated as a pedicle, and the whole cyst removed.

With regard to the cause of the suppuration in these cysts, the evidence is strongly in favour of the view that in the great majority of cases the suppurative change in the ovary is secondary to purulent salpingitis. Of the thirty cases in which suppurating ovarian cysts were discovered, it will be seen from the annexed table that active puru-

TABLE IV.

Suppurating Ovarian Cyst (intra-pelvic).

Directly communicating with suppurating Fallopian tube...	8
Adherent to suppurating Fallopian tube	5
Adherent to inflamed Fallopian tube	12
Adherent to ulcerated vermiform appendix	1
Source of infection undetermined	4
	30

D

lent salpingitis was found in thirteen and chronic salpingitis in twelve. In the latter the conditions of the parts around showed that the tubal inflammation—though now chronic and comparatively quiescent—had originally been acute and severe. The fire had here, as it were, died out, though, in the neighbouring structures, to which the flames had extended, it was still raging. Thus, 83 per cent. of the cases of suppurating ovarian cyst were associated with salpingitis. If it be asked why may not the tubal disease be secondary and the ovarian primary, I reply that if that had been the case one would have expected the mucous lining of the tube to be the last part affected, and the least; in other words, one would have expected evidence that the inflammation of the tube had passed from the peritoneal coat inwards rather than from the mucous coat outwards. In eight instances the suppurating tube and the suppurating ovarian cyst were, at the time of the operation, in direct communication, owing to ulceration of the cyst wall and perforation into the adherent tube. Whether the cystic change in the ovary ever itself begins as an indirect result of inflammatory changes involving the outer coat of the ovary, I cannot say. But certainly, in the majority of cases, it is much more likely that there already existed a small cyst, the contents of which became infected from the adjacent tube and underwent suppuration; and that, under these circumstances, the cyst grew so rapidly as to be easily mistaken for a large abscess in process of formation. Of the five remaining cases of suppurating ovarian cyst, in one the cyst had evidently become infected from a detached vermiform appendix; whilst in the remaining four the source of the suppuration was not discovered. It is, of course, possible that in these the cysts became infected from their propinquity to the rectum.

To the remaining cases of intra-pelvic suppuration enumerated in the table, I propose only to allude very briefly. The subject of tubercular disease of the uterine appendages, of which there are seven cases on the list, is one of extreme interest, but is much too large to be adequately dealt with on the present occasion. I would merely say, in passing, that when the disease is limited to these parts, or when the only other manifestation of the disease is miliary tubercle of the peritoneum, removal of the diseased appendages is not only a justifiable operation, but is frequently attended with the most satisfactory results. The female pelvis is one of the situations where we are

happily able, when the condition has been discovered in time, to rid our patients of the disease before it has become a focus of general tubercular infection.

The case in which an intra-pelvic suppuration was found to be due to a diseased appendix vermiformis does not, of course, properly come within my province as a gynaecologist. It happens, however, that the acute attack of peritonitis for which the patient was admitted occurred a few days after parturition, and was naturally thought to be connected with that process. As it is only under exceptional circumstances that such cases come under my care, it is obvious that my individual experience affords no guide to a correct estimate of their relative frequency.

The six cases in which the seat of the suppuration was not definitely made out, occurred in the earlier part of my work, when, having not yet attained the boldness that comes of experience, I was content to empty and drain any deep-seated collection of pus in the pelvis without attempting to separate and remove the suppurating organ.

Fistula as a complication of pelvic suppuration.

I propose to devote the remainder of the time at my disposal to a consideration of those cases in which collections of pus within the pelvis have burst into the rectum, the genital canal, or the bladder, and in which, the opening being insufficient and the evacuation of the pus consequently incomplete, a sinus remains through which there is a constant or intermittent purulent discharge. From amongst a number of such cases that have come under my observation, I propose to relate a single example of each of the main varieties of this form of fistula, beginning with the most common one, that, namely, which occurs as the result of the bursting of a collection of pus into the rectum.

Case 1. A woman, aged 35, who had been in good health all her life, and was entirely unaware that she had a tumour of any kind, was attended in the maternity department of St. Thomas's Hospital in her eighth confinement. The presentation was an occipito-posterior, and the head was arrested above the brim. After failure with the forceps, delivery was easily effected by version. On the sixth day the temperature, which had, hitherto, not exceeded 99.2° F. rose to 100.2° F. The patient had no pain of any moment until the seventeenth day, when she complained of a dull, aching pain in the lower part of the back and

outer part of the left thigh. There had been no rigor, or sickness, or headache, or constipation, but the patient was losing flesh, her appetite was poor, and she complained of much thirst. On the nineteenth day there was noticed for the first time some swelling in the abdomen. The temperature, which had been gradually rising for a fortnight, was now 103° F. Accordingly, on the following, *i.e.*, on the twentieth day, the patient was admitted into the hospital.

The note on admission describes her as thin, pale and sallow. The chest sounds were normal. The abdomen was irregularly distended. The uterus reached to within three inches of the umbilicus, and was pushed forwards so as to form an obvious projection of the anterior abdominal wall. Both uterus and bladder were drawn upwards so as to lie entirely above the pubes. On deep palpation, behind and above the rounded upper margin of the uterus, could be felt a swelling like a tense cyst.

On vaginal examination a centrally-situated swelling, continuous with that felt behind the uterus in the abdomen, was found to occupy the hollow of the sacrum, distending the pouch of Douglas. The swelling was of unequal consistence, and in one part gave a sense of fluctuation.

The temperature ranged from 99° Fahr. in the morning to 101° Fahr. in the evening. The urine was healthy.

It was thought at first the swelling might be a hæmatocele, but when the patient had been nearly three weeks under observation, a large quantity of very foetid pus was passed by the rectum.

The temperature at once fell to normal, and next day there was a marked diminution in the size of the swelling. Pus continued to be passed with each evacuation for four days. After an interval of six days the temperature again rose, and the size of the abdominal swelling became evidently increased. Next day there was a further escape of pus from the rectum, and this continued without diminishing the temperature for four days, when abdominal section was performed. The swelling proved to be a suppurating dermoid cyst of the right ovary. The cyst was everywhere adherent, and the process of separating and removing it was one of extreme difficulty, occupying two hours and a half. Notwithstanding the utmost care some of the foetid contents of the cyst escaped into the abdominal cavity. The peritoneum was subsequently flushed with hot boracic acid solution,

and a drainage-tube was inserted at the lower angle of the incision.

The cyst contained a compact mass of hair 4 inches by 3 inches, some pieces of bone, and, in a separate loculus, 28 fl. oz. of thick yellow fluid which solidified on cooling. A direct communication existed between the cyst and the right Fallopian tube, which was nine inches in length and greatly thickened, and was in a state of acute suppurative inflammation. The cyst had also opened by ulceration into the rectum.

Convalescence was somewhat protracted, but no pus was again seen in the evacuations, and none of the contents of the bowel ever escaped through the wound. The patient left the hospital well in ten weeks. It is now upwards of two years since the operation, and the patient, whom I saw a fortnight ago, remains perfectly well.

The next case is one in which a sinus existed in the vault of the vagina for eight months, and was found at the operation to communicate with one of a series of small suppurating cysts of the right ovary. It is of special interest (1) as being a typical example of the cases that have continually been described by observers who do not operate as instances of cellutic abscess bursting into the vagina; and (2) as affording an unanswerable argument in favour of dealing with such cases by abdominal section rather than by enlarging the sinus from below. The case has already been published in detail,* and I shall therefore be content with giving it here in abstract.

Case 2. A young married woman, æt. 22, was delivered of her first and only child at the age of 19. For twelve months after the labour she suffered from constant pain in the right iliac region. She then remained well for some few months, when the pain returned, and one day, eight months before admission, a sudden discharge of thick, yellow, foetid matter took place from the vagina.

The discharge had continued, with short intervals, during the whole of the eight months, but, since the first day, had not been offensive. Acute symptoms set in a week before her admission. On vaginal examination the uterus was found in normal position, the cervix fixed. The pouch of Douglas was occupied by a hard rounded mass, extending further to the right side than to the left. The vaginal roof on the right side was slightly depressed. There was dense hardness in the tissues

* "Obst. Soc. Trans." vol. xxxiv for 1892, p. 387.

at the posterior vaginal reflection and immediately in front of the cervix. At the upper part of the posterior vaginal wall was a small opening, the size of a pea, with indurated margins.

The diagnosis was vaginal fistula communicating with the inflamed and suppurating right uterine appendages.

Abdominal section was performed. The pelvic viscera were densely matted by old adhesions. The broad ligaments were hard, rigid and thickened. A soft oblong mass, consisting of the right ovary and Fallopian tube, dipped down into Douglas's pouch, where its dense adhesions were separated with difficulty. On examination, the diseased ovary was found to measure $2\frac{1}{2}$ in. by $1\frac{3}{4}$ in., and to consist on section, of a number of inflamed cysts, many of them full of pus, and all with hyperæmic walls. An opening, large enough to admit a goose-quill, and surrounded by granulation tissue, was found on that part of the surface of the ovary which had lain most deeply in the pelvis. This opening communicated directly with one of the abscess cavities in the substance of the ovary, and pus was seen exuding from it. The right tube was attached to the ovary, and was elongated. On section its canal was found empty, and its lining membrane healthy.

Five weeks after the operation, the patient left the hospital well. There had been no further purulent discharge from the vagina. Ten months later no swelling could be detected, on vaginal examination, on either side of the pelvis. Menstruation was regular, and, except for occasional attacks of pelvic pain, unaccompanied with rise of temperature, the patient felt well and strong.

Had the treatment here consisted of enlarging the sinus in the posterior wall of the vagina, only one cyst would have been laid open, and the patient would have been left with several others to give trouble later on.

The attacks of pain described by the patient as having occurred at intervals since the operation, are probably to be explained by intestinal or omental adhesions at the site of operation.

The next case, like the first that I related, proved to be a suppurating dermoid of the ovary, situated deeply in the pelvis, but instead of ulcerating through into the rectum, it opened into the cervix uteri, resulting in a fistula in that situation. I published the case in full in a paper in the 17th volume of the St. Thomas's Hospital Reports and shall here merely give an abstract of it.

Case 3. A married woman, æt. 36, was admitted into hospital nineteen weeks after her fourth confinement. Her first three labours had been natural; the last one had been very difficult, delivery having been accomplished by the use of forceps, and then only after prolonged effort. She became feverish soon afterwards, and had much vomiting. The lochia were arrested. A fortnight after the labour a purulent discharge took place from the vagina; this went on for a few days, when, the flow not being very free, an opening felt on making a vaginal examination was enlarged by the medical attendant. The purulent discharge continued for thirteen weeks, gradually diminishing in quantity; it then suddenly became more profuse and very offensive, and the opening was again enlarged. Not long after this a quantity of horribly offensive pultaceous material passed, together with a quantity of hair three or four inches in length. This offensive discharge went on up to the time of her admission. The patient had endeavoured to occupy herself in the house, but found that she was becoming thinner and weaker, and that she was never safe from a sudden outburst of the ill-smelling discharge. An abdominal swelling had been noticed for the first time about six weeks after the confinement.

On admission, the patient, who was sent in as a case of pelvic cellulitis, was pale, wasted, and very ill. She had a rounded tumour in the right iliac region, reaching to the level of the umbilicus. The posterior fornix of the vagina was obliterated, the finger, on entering the vagina, passing directly into the cervix uteri. On the inner and posterior wall of the cervix could be felt a lacerated depression, such as would be caused by the passage of a moderate-sized trocar. No opening was discoverable in the vaginal wall. There was a hard, rounded mass in Douglas's pouch, depressing the vaginal roof.

On opening the abdomen, the swelling proved, of course, to be a suppurating dermoid of the ovary. The tumour was carefully and with much difficulty detached all round, a quantity of highly offensive pus flowing from the vagina during the manipulation. Before separating the adhesions in the neighbourhood of the perforation, the pedicle was divided, in order that the tumour might, after separation, be lifted quickly out and the inevitable soiling of the pelvis from the escape of the cyst contents reduced to a minimum. As the tumour was removed, a gush of highly offensive gas escaped with a distinct whiz.

The peritoneal cavity was repeatedly douched, and after the abdomen had been closed and the patient had rallied a little, the vagina was thoroughly douched with hot boracic lotion.

The convalescence was somewhat delayed by suppuration within the pelvis, but the patient was able to sit up in bed in three weeks, and at the end of six weeks she left the hospital well. Five weeks later she presented herself, looking remarkably well.

This case, like the first, is an instance of suppurative inflammation of an unsuspected ovarian cyst, closely following a difficult labour. It is easy to understand how readily the true nature of such a case may be overlooked. The probability is that, in this latter case, some laceration of the posterior wall of the cervix took place during delivery, opening up a channel by which septic infection could easily reach the tumour, which had, no doubt, been rendered specially susceptible owing to the bruising it had recently undergone.

The next and last case that I shall relate is, to my mind, the most interesting of all. It was sent into the hospital as a case of pelvic cellulitis following influenza, and complicated with abscess which had burst into the bladder.

Case 4. The patient, a woman, æt. 32, had been married nine years, but had never been pregnant. She had had an attack of inflammation in the right side of the lower part of the abdomen three years previously, the attack having been preceded for a few days by a yellow vaginal discharge. After this, she had remained well until six months before her admission, when she had what was supposed to be an attack of influenza, with shivering and perspiration, but no pain. She kept her bed for a fortnight, and then went into the country. Whilst there she noticed some thick yellow matter in the urine, which had continued to appear ever since, that is, for a period of five months, the daily quantity being estimated by the medical attendant to average about $\frac{1}{2}$ fl. oz. There had been frequency of micturition, but no dysuria. The general health had been but little affected. Menstruation had been regular. She had suffered pain from time to time in the right iliac region, but this had never been very severe.

On examination under anæsthesia, the uterus was found of normal length, the canal directed to the left. On the right side of the pelvis a large, hard, uneven, roughly globular mass was felt, depressing the vaginal roof, and extending

upwards to the level of the anterior superior iliac spine. A sound introduced into the bladder was prevented by the swelling from passing beyond a very short distance in the middle line, but passed easily to right and to left of it posteriorly. The urine contained a varying quantity of pus.

It was evident that there was chronic suppuration of the uterine appendages (probably, from the size of the swelling, of both tube and ovary), and that there was a sinus communicating with the bladder. I had not yet had occasion to operate upon a case of this kind, and although I saw no other satisfactory way of dealing with it, I undertook the operation with considerable misgiving. However, on the 8th of December of last year, I opened the abdomen. After separating and drawing aside the omentum which completely concealed the contents of the pelvis, the mass was easily seen occupying the right and central portions of the pelvis, deep down. It was covered by peritoneum, and there did not for some time seem to be any possibility of finding a break in the apparent continuity of the covering of the mass with the peritoneum of the pelvic walls. At length it was found possible to insinuate a finger behind and to the right of the uterus and so to commence the work of separation.

The thickened right broad ligament had been drawn over the anterior surface of the diseased parts and dipped down superiorly where it was adherent to omentum and pelvic wall. These adhesions having been separated, access was obtained to the mass beneath, and, with the fingers of an assistant in the rectum to serve as guide, separation was slowly and carefully effected. The mass when removed proved to consist of the inflamed right Fallopian tube, and a suppurating cystic ovary three inches in diameter. The firmest adhesion was in the neighbourhood of the fibrilated end of the tube. When this was separated a quantity of soft inflammatory debris was set free. The tube was dilated, and its mucous membrane congested and œdematous; no pus was present in its canal. The ovary was removed without rupture. Its surface was convoluted like that of a tomato. On its under-surface was a small opening, from which blood-stained purulent fluid oozed on pressure, and which probably represented the aperture of communication with the bladder. On section it was seen to contain two separate cysts, each of them filled with purulent fluid of a brick-red colour, and without ill odour. One of the cysts

was surrounded by indurated tissue $\frac{1}{2}$ in. thick. The bladder was deep down, quite out of sight and out of reach, so that any attempt to close the opening in its wall was entirely out of the question. No urine escaped, and very little pus appeared to have exuded into the pelvis during the operation. It was thought prudent, however, to wash out the pelvis well with hot boracic solution. The uterus had by this time moved to its normal position in the middle line, and perched on the fundus was the normal and adherent left ovary. The left tube was adherent, but apparently free from disease. On passing a catheter a drachm or two of blood-stained purulent fluid escaped from the bladder. A drainage tube having been inserted the incision was now closed.

The urine drawn off up to midnight contained pus and blood. The bladder was at first emptied by catheter every two hours to prevent distension. Next day the interval was prolonged to three hours, and on the fourth day to four hours. The discharge through the drainage-tube at the first dressing was turbid, and contained leucocytes; it then became chiefly serous, but later it again became purulent, though never urinous. There was some cystitis, with alkaline urine, from the fourth to the tenth day, when the urine became natural. The patient was able to sit up on the sixteenth day, and left the hospital, looking stout and well, at the end of two months. Notwithstanding that the general condition was excellent, there continued to be a little suppuration from the lower angle of the wound for six months, when the sinus finally closed. The patient has presented herself since from time to time, and when I last saw her, a month ago, she was in perfect health, and menstruating regularly.

I have narrated these cases with two objects specially in view. The first was to show that when pelvic suppuration is complicated by internal fistulæ, the suppuration is not cellulitic, however much it may simulate it, but is due to intra-pelvic disease that can only be properly dealt with by abdominal section. The second object I had in my mind was to show the feasibility, even in the most unpromising cases, of complete removal of the disease, and to urge the superiority of that method of treatment over the mere emptying and draining of the suppurating cavity and the stitching of the edges of the sac to the abdominal incision.

I am painfully conscious that in offering the foregoing remarks to my professional brethren in Bir-

mingham and the Midlands, I am addressing many who have had a far larger experience than I in the treatment of the diseases to which they refer, and before whom it would be much more fitting that I should appear in the capacity of learner than of teacher. Nevertheless, I trust they will appreciate my effort to prove to them that the example they have set, in endeavouring, in spite of much opposition, to advance the department of medicine with which I am more especially identified, has not been altogether lost, even upon the benighted Londoner.

* NEPHRECTOMY FOR HYDRONEPHROSIS.

By J. BLAND SUTTON,
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THE conditions of the kidney known as hydro- and pyonephrosis have, during the last ten years, become of great interest to surgeons, and the methods of dealing with them have been so improved that nephrectomy for hydronephrosis has become as safe as an uncomplicated ovariectomy.

Having had several opportunities of operating upon some unusual cases of cystic kidneys, I thought it might be of interest to bring the matter under the notice of this Society, in order to demonstrate the comparative safety of nephrectomy for hydronephrosis when carried out on certain definite lines.

Before dealing with the purely surgical side of the subject it will be necessary to discuss some points connected with its pathology. That by far the greater number of hydronephroses are due to some easily demonstrable obstruction in the urethra, bladder, ureter, or pelvis of the kidney is indisputable; hence it is customary to describe a hydronephrosis as a *retention-cyst*. It is, however, important to bear in mind that hindrance to the free escape of urine must be either *incomplete*, or if *complete only temporary* to produce hydronephrosis. When the ureter is completely and permanently plugged the kidney rapidly atrophies. Experimental facts support the clinical evidence in this particular.

When the obstruction is partial, or temporarily

* A Paper read before the Medical Society, London, Nov. 13, 1893.

complete and frequently recurs, its effect upon the renal pelvis is very striking.

The most startling fact a study of my cases and specimens bring out is this: Many of the largest hydronephroses are those in which no obstruction could be demonstrated, and the histories of the cases fail to throw any light upon the causes inducing the hydronephrosis. For a long time I felt that this apparent anomaly could be explained by abnormal movements of the kidneys producing kinks in the ureters; this explanation, though satisfactory occasionally, is in some cases totally inadmissible. It is also necessary to mention that the largest hydronephroses are usually those which are subject to intermission. All hydronephroses which form a tumour capable of being detected clinically intermit, but the phrase "intermitting hydronephrosis" is exclusively reserved for those cases in which great diminution, and in some instances temporary disappearance of the swelling occurs.

I once had an opportunity of actually witnessing the phenomenon of intermission whilst performing an abdominal section: the case is also of great interest as the patient was the victim of bilateral hydronephrosis, and the cysts used to intermit alternately.

This woman, 42 years old, was placed under my care in 1890 on account of an abdominal tumour. On examining her I found a very large swelling on the left side and a smaller but very similar one on the right, furnishing the physical signs of renal tumours. Seven days later the patient was admitted into the hospital; at this time nothing abnormal could be made out in the right loin, but the left side of the belly was occupied by a large cyst reaching into the pelvis. The uterus was occupied by a myoma the size of a fist. A consultation was held upon the case, and the physicians present at the conference were inclined to regard the swelling as an ovarian cyst. To clear up the doubt I decided to explore the abdomen. In due course the patient was anesthetized, and whilst engaged incising the belly-wall I saw the swelling gradually diminish in size, an alteration also noticed by the bystanders. On opening the peritoneal cavity the bladder was found distended with urine, and as it had been emptied by catheter just before the exhibition of ether, it was clear that the sudden diminution in the size of the tumour was due to the escape of urine from the enormously dilated pelvis of the left kidney. I took the opportunity to

examine the right kidney, and it appeared natural in size and consistence. The uterus was acutely retroflexed by a myoma, the size of a fist, in its fundus. The patient was so upset by the operation, that further interference was for the time injudicious. The wound quickly cicatrized, and the patient left the hospital three weeks later (July 26th). In eighteen days she returned very ill, and in suffering with a very large tumour in the *right* loin, that in the left having subsided to such an extent, that it was scarcely perceptible to the fingers on manipulation. On August 21st I exposed the right kidney through an incision in the loin and evacuated thirty ounces of thick pus, and then removed the remnant of the kidney in spite of the hydronephrotic condition of its companion. A rapid recovery was the consequence. I naturally felt anxious as to the subsequent fate of the left kidney.

The patient continued in excellent health for more than two years; she then began to experience difficulty in micturition at the menstrual period, and stated that the swelling in the left side was larger during menstruation, an observation which seemed confirmed by the fact that the uterine tumour had trebled its size since the patient first came under observation. The clinical evidence appeared to point so conclusively to the myoma being the source of trouble by pressing on the ureter, that I decided, after consulting with my colleagues, to anticipate the menopause by removing the ovaries. This measure was carried out. For about eight days the patient's condition was very promising indeed; then uræmic symptoms became prominent, and the temperature fell to 96°, and remained at this point in spite of active efforts to make it rise, and on the twelfth day after the operation the woman died.

At the *post-mortem* examination the left kidney was found transformed into a large sac without any trace of renal tissue visible to the eye. The ureter was of normal size, and the uterine tumour had shrunk to the size of a fowl's egg.

These facts made it clear that the myoma was not responsible for the hydronephrosis, and the cause in this case remains unexplained.

In this unusual case, the fact that both kidneys were cystic enabled me at all times to satisfy myself of the existence of a tumour on one side or the other, notwithstanding the fact that both tumours intermitted, but the circumstance that the intermission was alternate simplified in a large

measure the clinical aspect of the case. When intermission occurs in a unilateral hydronephrosis, the occasional sudden disappearance of a tumour will often cause a great deal of perplexity in the mind of the observer. The following case will illustrate the diagnostic difficulties which sometimes arise :

In 1890 a tall well-proportioned single woman, 26 years of age, was placed under my care for supposed ovarian disease. A careful examination under chloroform made it clear that the pelvic organs were healthy. Three months later the patient was again sent to me for the purpose of oöphorectomy. After keeping the case in bed for a week, and re-examining her, I made out some indefinite resistance in the right loin, and came to the conclusion that the trouble was more probably due to renal than to ovarian disease. Two years later I was informed that this woman had been in University College Hospital, where the surgeon discovered a large renal swelling and had arranged to explore it, but a few hours before the time fixed for the operation the tumour vanished. Shortly after this the patient came to me again, and on examining her I made out a very large swelling in the right lumbar region. In August, 1892, she was admitted into the hospital, and at a consultation with the oldest and wisest of my colleagues, the most extraordinary opinions were expressed,—one even doubted the existence of a tumour. I gained little by this conference, except an increased responsibility. To ease the doubts, the abdomen was opened in the middle line as for an ovariectomy. The right kidney was detected as a huge cyst: the ureter was traced into the pelvis, but I detected nothing abnormal in connection with it, the ovaries, tubes, uterus, or broad ligaments. The left kidney was natural. I closed the median incision, turned the patient into a lateral position, and removed the hydronephrosis, which had a capacity of nearly 100 ounces, through a wound in the loin. Recovery was rapid and complete.

Though the diagnostic exercise this cyst caused was great, yet I venture to suggest that the pathological difficulties are even greater, for there is no evidence of partially plugged ureter, or pressure on this duct from a pelvic tumour, and the connections of the cyst to the surrounding parts prevented rotation or any vertical excursions capable of temporarily obstructing the ureter, yet in spite of this, the kidney became converted into an intermitting hydronephrosis.

An unusual example of unilateral hydronephrosis associated with great dilatation of the ureter is represented in fig. 1. This specimen was taken after death from an old man. The closest ex-

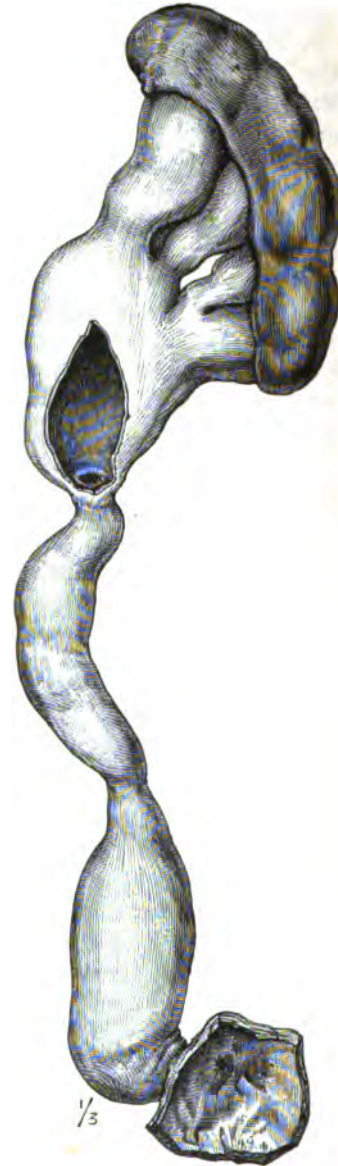


FIG. 1. A hydronephrosis associated with great dilatation of the ureter. (Museum, Middlesex Hospital.)

amination of the ureter and bladder failed to detect an obstruction. During life there were no symptoms indicating renal disease, and the *post-mortem* examination was undertaken for the purpose of finding the cause of an inexplicable illness.

The silent way in which the gradual dilatation of the renal pelvis, infundibula, and calyces destroys a kidney is very extraordinary. When hydronephrosis is unilateral it rarely betrays itself until the tumour is very large; frequently the only trouble it causes is increased frequency of micturition.

When the hydronephrosis is bilateral the signs are often in abeyance until the amount of renal capital is reduced to the minimum amount capable of meeting the ordinary demands of the individual: directly there is an extra call, then the low amount of available renal tissue becomes alarmingly manifest, and the patient dies. The suddenness with which renal insolvency sometimes declares itself is evident in the following case:—

In June, 1893, a man of 26 years was placed under my care, under the supposition that he was suffering from acute intestinal obstruction. The patient was too ill to give a reliable account of himself, and his mother could tell us no more than that she found him very ill in his lodgings. The belly walls were rigid but not much distended: the flanks and loins were dull: the patient ejected a quantity of foul-smelling vomit, and had passed no urine for eighteen hours: the bladder was not distended, and so far as could be ascertained, there had been no action of the bowels for some days. The rectum was empty; pulse feeble and running; temperature 96°. I deemed it unwise to interfere with the case, but some of my colleagues thought the evidence so strongly indicated intestinal obstruction that he ought to be submitted to an exploratory incision. I grudgingly consented, and on making an incision in the left linea semilunaris came upon a large hydronephrosis. This was stitched to the wall and drained. The right kidney was in a similar condition. The man died twelve hours later. At the *post-mortem* examination, the kidneys were merely fibrous sacs the hydronephrosis being due to a vesical calculus the size of a bantam's egg. From an inquiry made among the man's relatives we found that he had never complained of any illness, and had continued to work until a few days before his admission to the hospital.

Although a hydronephrosis continues its course in silence it is almost certain to be made manifest when it suppurates, and my observations among the quick and the dead have taught me that this is one of the greatest dangers to which individuals with unilateral hydronephrosis are liable.

It is necessary to draw a distinction between

pyonephrosis and suppurating hydronephrosis. In the case of a pyonephrosis the lesion is inflammatory from the outset, whether it start primarily in the kidney or spreads to this gland from the bladder, ureter, or elsewhere. A suppurating hydronephrosis is comparable to a suppurating ovarian cyst, requires similar treatment and furnishes incomparably better results. A primary pyonephrosis rarely attains a large size, and as it is very frequently associated with calculus it demands nephro-lithotomy or nephrotomy rather than nephrectomy.



FIG. 2. A suppurating hydronephrosis.
(Museum, Royal College of Surgeons.)

An example of suppurating hydronephrosis associated with calculus is represented in fig. 2. (It was successfully removed by Mr. Meredith.) The strait formed by the junction of the pelvis with the ureter lodges a peculiar hammer-shaped calculus; it is clear from its shape that it could not completely occlude the orifice of the ureter, but it would so hinder the free escape of urine as to produce hydronephrosis, which subsequently became a pyonephrosis. It, of course, does not necessarily follow that when a calculus is associated with a largely dilated pus-containing renal pelvis that the condition was primarily septic.

It is an interesting matter to inquire into the modes by which a hydronephrosis becomes septic. In very many cases there can be no doubt that the channel of infection is the ureter. The opinions formerly held that the healthy ureter allows no retrograde communication between bladder and renal pelvis is being gradually weakened; a dilated ureter certainly allows very free currents to pass from the bladder to the renal pelvis. There is, however, another source to be reckoned with—the colon. Whilst operating I have satisfied myself that a large hydronephrosis comes into very intimate relation with the ascending or the descending colon, and the walls of the cyst and the gut sometimes become so blended and thin as to permit osmosis of fluid from the bowel into the hydronephrosis and establish suppuration. Under such conditions the patient's sufferings are apt to be confounded with typhoid fever, a confusion which sometimes arises in connection with the suppuration of an ovarian cyst. In the early part of 1893 I had a striking example of this in a young Frenchwoman placed under my care by Dr. Beer. The patient had been treated many weeks, in a hospital in Brussels, for a supposed attack of typhoid fever, but at last she came in despair to some friends in England. Dr. Beer found a large swelling extending from the pelvis to the fifth rib on the left side; this swelling was dull front and back, there was a marked bulging in the ilio-costal space. The patient had a hectic temperature, and the urine contained a slight trace of pus. Immediately on her admission to the hospital I made an incision into the left ilio-costal space, and evacuated three pints of pus. A large cystic mass could be made out in the situation of the kidney, and through a small orifice on its posterior wall pus was leaking. On enlarging this aperture my finger entered a large sacculated kidney. Whilst my assistant was washing out the abscess cavity I gently enucleated the kidney from its capsule with my finger, tied the pedicle, and removed it. I have never seen a larger abscess cavity than was then exposed to view; the dome of the diaphragm formed a sort of apse at the upper end, and the ilio-psoas, ragged and torn, limited it below; the nerves of the lumbar plexus crossed the dorsal wall of the cavity like fiddle strings. The woman made an admirable recovery. Nothing was found to account for the dilatation of the kidney; the descending colon was incorporated in the inflammatory mass representing the renal capsule. In this case it is

very probable that this woman had a hydronephrosis, which became fouled from the colon, and the signs consequent on the establishment of suppuration in the sac were misinterpreted. In due course pus leaked into the circum-renal tissue, and set up a perinephritic abscess of unusual dimensions.

Diagnosis. The clinical recognition of a suppurating hydronephrosis is beset with far less difficulty than a simple hydronephrosis. The presence of a tumour in the loin accompanied by an elevated temperature and the escape of pus with the urine are well-known signs. A most important point in diagnosis is to distinguish between renal tuberculosis, pyonephrosis due primarily to calculus, and a suppurating hydronephrosis. Clinical experience enables us to do this with a fair amount of accuracy, and the differentiation is made rather by intuition, the offspring of experience, than by methods capable of being expressed in written rules.

Very large hydronephroses have been many times mistaken for ovarian cysts; also for pancreatic cysts, and tumours of the liver.

When of moderate size they are apt to be confounded with hydro- and pycholecysts, scybala in the colon, omental tumours, hydatid cysts, hepatic tumours, and pedunculated processes of the liver.

In framing a diagnosis never resort to what is called "aspiration for clinical purposes." This measure must be absolutely condemned, for it is useless, often misleads, and is sometimes the source of much danger. To thrust a trocar and cannula into an abdominal cyst, or tumour of any kind, for diagnostic purposes, is bad practice; it is inadmissible in the case of ovarian cysts, and is equally reprehensible with renal tumours.

Treatment. When a hydronephrosis is so large as to be clinically appreciable, it almost invariably causes the patient inconvenience, in a large number of cases is a source of much suffering, and in exceptional cases produces intense agony. The attacks of pain are probably due to variations in pressure of the fluid in the cyst, for, as has already been mentioned, almost all large hydronephroses intermit, more or less.

Another symptom, and one which is sometimes very distressing, is frequent micturition, unassociated with disease of the bladder or urethra. This sign is not so well appreciated as it should be. I have seen several cases in which female patients have had their bladders explored when complaining of frequent micturition, without effect, when a careful

examination of the abdomen has led to the detection of cystic kidneys.

When the surgeon has satisfied himself that an individual has a large sacculated kidney, and the fellow gland is in good condition and working properly, the disorganized kidney should be removed, and especially if it has suppurated. When both kidneys are hydronephrotic, the removal of one may end in disaster. I have in the course of this paper however, mentioned one case in which a suppurating hydronephrosis was successfully removed when the opposite kidney was known to be seriously disorganized. In the face of this grave complication, incision and drainage (nephrotomy) is the wiser course. In such cases it is well to remember that the power of secreting urine is of more importance to the individual than the capability of thinking.

Nephrectomy. The method of removing hydronephrotic and pyonephrotic kidneys which has given me the most satisfactory results is the following:—

The peritoneal cavity is opened by an incision in the linea semilunaris and the nature of the tumour ascertained; through this incision it is a very simple proceeding to ascertain the condition of the opposite kidney. Exceptionally, where the diagnosis lies between an ovarian cyst or a hydronephrosis, I have made the exploratory incision in the middle line as for ovariectomy. If all is satisfactory the abdominal wound is closed, and the patient arranged in a lateral position, and the cyst exposed through an incision in the ilio-costal space. No attempt is made to enucleate the tumour until the finger is well within the capsule; this is a cardinal point in the operation. It is due to ignorance of, or disregard to, this simple fact, that such accidents as tears in the peritoneum, colon, or diaphragm occur. When the cyst is well exposed it is tapped with a trocar and cannula, and its contents allowed to escape. The cyst wall is then gently enucleated and drawn through the opening in the loin. In the process of enucleation the large dilated ureter is often isolated; under these conditions it should be caught with forceps, transfixed, emptied of urine, and ligatured. When the cyst is freed from its parietal connections, the pedicle, consisting of renal artery, vein, nerves and ureter, unless this has been separately secured, is transfixed and secured with silk ligatures as in ovariectomy. The pedicle should be transfixed as close to the cyst as possible, especially when removing a right kidney. *Never clamp the pedicle*

with large forceps before securing the ligature.

The advantage of transfixing the pedicle near the kidney, is due to the fact that instead of tying the short renal artery, the ligature embraces the numerous branches of the vessels into which it subdivides as it enters the sinus of the kidney. After securing the pedicle the ligatures are shortened, and the cyst cut away with scissors; any parietal vessels that may be bleeding are secured, a large drainage is inserted, and the wound closed by sutures. In closing the wound it is desirable to join the cut edges of the muscles and especially the fascia, with buried gut-sutures. The skin is brought together with superficial silk sutures in the usual manner.

Before suturing the wound the surgeon ascertains that *the number of sponges is correct.*

In this sketch of the method of performing nephrectomy through the ilio-costal space, and therefore behind the peritoneum, I followed closely the teaching of my surgical colleague, Mr. Henry Morris, who has done so much to advance the surgery of the kidney.* One of the most important points in performing nephrectomy by the lumbar route is, *never to begin to detach the kidney until the finger is well within the capsule.* Neglect this precaution then the operator is very liable to the following accidents: (1) to tear the peritoneum; (2) push the finger through the diaphragm, and open the pleural cavity; (3) to injure the colon.

In securing the pedicle it is advisable to keep close to the kidney; non-observance of this precaution in removing the right kidney has been accompanied by such serious accidents as: (1) transfixion of the inferior vena cava; (2) inclusion of a loop of the duodenum in the pedicle.

In securing the pedicle the ligature must be tied very tightly, as it is more liable to slip than the stump of an ovarian tumour. Such an accident is certain to entail loss of life if it happen after the patient is returned to bed.

Whilst applying the ligature do not drag on the kidney; when the parts are made tense a glance at the patient's face will convince you that the interference with the solar plexus reduces the boundary between life and death to very slender proportions.

* The removal of a hydronephrosis through the loin, and the converse plan of removing a solid renal tumour through the belly, aided by a preliminary incision in the loin, as described in this Journal, Nov. 1, 1893, p. 8, are so identified with the teaching of this surgeon that they should be spoken of as Morris' methods.

Convalescence may be interfered with by (1) suppression of urine; (2) hæmorrhage; (3) sloughing of the pedicle; (4) thrombosis of the vena cava; (5) fæcal fistula; (6) septic troubles; (7) sponges or plugs of cotton wool in the wound; (8) pleurisy.

Lastly, success in nephrectomy demands, *decision*, *celerity*, and *gentleness*, qualities which can only be acquired by constant familiarity with renal operations.

One of the most important points in connection with hydronephrosis which requires elucidation is the fate of an individual who has been submitted to nephrectomy. At present, judging from the facts at my disposal, the outlook is very re-assuring, but its satisfactory consideration must be delayed for a future occasion.

ETHER AND CHLOROFORM CLINICALLY COMPARED.

A Lecture delivered at the London Hospital on Friday,
Nov. 10, 1893, by

FREDERIC HEWITT, M.A., M.D. Oantab.,
Anæsthetist and Instructor in Anæsthetics at the Hospital.

GENTLEMEN,—I propose on the present occasion to direct your attention to the clinical differences which exist between the two important anæsthetics, ether and chloroform. Let me say, at the outset, that in discussing a subject of this nature it is particularly desirable that we should not wander from the highway of fact into the by-roads of theory and opinion. You have witnessed numerous administrations both of ether and of chloroform in the operating theatre; I now ask you to analyse and compare the two sets of phenomena. In this way I hope to place the ether *versus* chloroform question before you in a practical light. By recognising the merits and drawbacks of ether on the one hand, and of chloroform on the other, we shall find ourselves in a position to formulate certain conclusions which may be of value to us in our future practice.

It would hardly be fair, nor would it serve any very useful purpose, to institute a comparison between ether and chloroform administered in precisely the same manner. Experience has shown that in order to produce and keep up a satisfactory form of chloroform anæsthesia, this anæsthetic

should be given freely diluted with air; whereas ether anæsthesia is best secured and maintained, at all events in most instances, by a degree of air-limitation appropriate to the particular case. We must therefore compare the open method of giving chloroform with the close method of giving ether—the Skinner's mask, the square of lint, or the corner of a towel, on the one hand, with Clover's or Ormsby's ether inhaler on the other. I do not say that there are not cases in which ether should be administered, like chloroform, with a plentiful supply of air; such cases do occasionally come before us, though they are highly exceptional. In the ordinary run of cases, well-regulated air-limitation (for the introduction of which in ether-giving we are indebted to Clover) is not only most useful to the administrator, but beneficial to the patient. It renders rapid anæsthesia possible; it prevents excitement and struggling which might otherwise arise; and it lessens the amount of ether used. The restriction of the air supply in administering ether renders this anæsthetic as manageable, as rapidly acting, and as powerful in its effects as chloroform itself.

It must be admitted that ether compares rather unfavourably with chloroform, at all events from the patient's point of view, in the initial stage of the administration. But it is to be remembered that by *properly* administering ether from a Clover's inhaler the irritant qualities of the vapour may be greatly lessened. The to-and-fro breathing of a limited quantity of air for half a minute or so before ether is admitted to the air current, brings about such an alteration in the perceptive faculties of the patient that the gradual admission of the ether vapour is hardly noticed. In fact, when ether is thus skilfully given, the patient certainly suffers less discomfort than when chloroform is recklessly poured upon a towel or sponge, and a too concentrated vapour presented to him. Time will not allow of my following up this branch of the subject further, but I may remind you that we have in nitrous oxide and in the A. C. E. mixture two agents which are particularly suitable for administering just before ether with the object of avoiding the discomforts to which I have alluded. When ether is to be given from the commencement there is no better inhaler than Clover's, because the strength of vapour can be gradually increased. But when nitrous oxide or the A. C. E. mixture has been employed to induce anæsthesia, ether may be advantageously given

by means of Ormsby's apparatus, and the anæsthesia will be found to be of a particularly satisfactory type.

Those of you who have watched the administration of anæsthetics in the room provided for the purpose will have observed that patients who are anæsthetised by means of Clover's ether inhaler properly used, very rarely become excited or struggle; whereas those to whom chloroform is given not infrequently gesticulate, grow noisy, and even become so violent that they have to be held. Muscular men of nervous temperament or alcoholic habits are particularly prone to give trouble in this direction. But when ether is properly given—and I may remind you that when administering this anæsthetic to such subjects it is necessary to practise air-limitation to a considerable degree, and to admit large quantities of ether vapour as quickly as is compatible with free breathing—the help of by-standers is very rarely needed. The stage of excitement, rigidity, and struggling is of very great importance in connection with the inhalation of chloroform; for during this stage a large number of chloroform deaths have occurred in persons who were at the time of administration in excellent health. All of you must be familiar with the frequent announcements in the medical and lay press headed "Death under Chloroform." You will often find it stated that the patient was a labourer, a plate-layer, a miner, or someone who, from the nature of his occupation, must have been in good health when the accident occurred which necessitated the use of an anæsthetic. Let us pause for a moment to consider the conditions present when a partially anæsthetised patient of vigorous build is thrown into a state of muscular spasm during the administration of an anæsthetic. Numerous muscles which are directly or indirectly concerned in maintaining free breathing share in the spasm; the air-way becomes temporarily occluded; the chloroform vapour present in the pulmonary passages continues to be absorbed by the circulating blood; in a word, mechanical asphyxia and a deepening narcosis rapidly become established. This state of things is met with under both anæsthetics, but it is far more hazardous to life under chloroform than under ether. With the last-named anæsthetic fatalities are extremely rare in connection with this asphyxial condition, except in the most exhausted and debilitated subjects.

When profound surgical anæsthesia has been produced, a careful comparison of the average

ether patient on the one hand with the average chloroform patient on the other, will reveal interesting and remarkable differences.

The respiration of the etherised patient is usually deeper, quicker, and noisier than that of the patient under chloroform; and more mucus and saliva are secreted. There is a greater tendency for swallowing and coughing to arise than when chloroform is employed. But the exaggerated breathing of ether, although inconvenient in many cases, is, as a rule, distinctly advantageous. In the first place it is an excellent guide as to the depth of anæsthesia and the patient's general condition. The quiet, almost imperceptible respiration not infrequently met with under chloroform—a condition which has created a demand for delicately adjusted registers for detecting the passage of air to and from the lungs—rarely, if ever, complicates ether anæsthesia. In the next place, when anæsthetising patients who have to be so placed upon the operating table that free chest expansion is rendered difficult, the deeper and more vigorous respirations under ether are of singular advantage from the anæsthetist's point of view. In chronic cases of empyema, too, when the patient is exhausted, and feeble respiratory movement is to be guarded against, ether, cautiously given with plenty of air, is more suited to the case than any other anæsthetic. But let us not forget that cases occasionally occur in which the respiration under ether is too forcible for the successful performance of the operation. This may be so in abdominal surgery; and in such cases we should have recourse to chloroform. And ether may, in certain types of patients (*e.g.*, the very emphysematous, the asthmatic, the phthisical, the very young, and the very old) produce considerable distress in breathing, and in such cases we prefer to administer chloroform or a mixture containing this agent.

The circulation of the patient under ether, like the respiration which we have just considered, is of an exaggerated type. The pulse is quicker and fuller than normal, the venous system is engorged, and incised parts bleed freely. The pulse of a patient deeply under chloroform is usually either slower than normal or about the normal rate, whilst its fulness is somewhat impaired; the venous system is not so engorged as under ether; and there is less vascularity of incised parts. These circulatory differences between ether and chloroform render the latter anæsthetic a somewhat more

suitable one from the operator's point of view; and it must be admitted that when, as in certain cases, excessive vascularity would greatly interfere with the performance of some important operation, the choice of the anæsthetic should certainly fall upon chloroform. The venous engorgement under ether may constitute a formidable complication, especially in deeply-seated operations at the root of the neck. For example, you will all have fresh in your memory a recent case in which the patient was dyspnoic from the pressure of an innominate aneurysm upon the trachea. The operator tied the carotid and subclavian arteries. Now in such a case ether would have been out of the question. It was contra-indicated from the respiratory as well as from the circulatory side. It would have led to tumefaction of, and increased secretion of mucus from, the tracheal mucous membrane, and it would thus have further narrowed the already small chink through which air entered the lungs. But in addition to this it would have increased the general and more particularly the local venous engorgement of the parts, and rendered the operation more difficult of performance. In several other operations, such, for example, as those in which deep dissections for the removal of cervical glands are necessary, ether may seriously inconvenience the surgeon by the excessive vascularity of the parts. But, as you may readily imagine, the exaggerated cardiac action under ether stands us in good stead in numerous other operations in which the vascularity of incised parts makes little or no difference. In fact it is the stimulant effect upon the heart that is the chief factor which renders ether such a safe anæsthetic.

For example, when too large a quantity of ether or of chloroform has been given and the respiration shows signs of failure, we observe a marked difference between the circulation of the patient poisoned by ether and that of the patient poisoned by chloroform. In the ether patient the pulse is usually beating satisfactorily, or at all events it can be readily detected; whereas, with chloroform it is either extremely slow and feeble, or, more commonly, cannot be felt at all. These differences are most pronounced in persons who are in a moderately good state of health. When from some exhausting disease or some similar cause the circulation and general vital powers of the patient have been reduced to a very low ebb, an overdose of ether may have very much the same effect as an overdose of chloroform. This will be seen by

referring to the ether fatalities which are on record—fatalities which as a rule have taken place in utterly broken-down and exhausted subjects. The fact that an overdose of ether in a cachectic subject is liable to be attended by symptoms of circulatory depression, similar to those which usually mark the toxic phenomena of chloroform in a healthy person, seems to suggest that in the latter case there is a something which is wanting when ether is similarly given in toxic quantities to a patient in good health. Whether this something is the depressing influence of chloroform upon the heart itself does not now concern us. The fact remains that when patients die from an overdose of chloroform, the death is in the vast majority of cases a cardiac one. It may be that breathing ceases before the action of the heart—this I am ready to admit. But the patient dies because we cannot restore his circulation. Just as the circulatory phenomena of a patient poisoned by ether differ from those of a patient poisoned by chloroform, so do the circulatory phenomena which attend the suspended breathing of imperfect anæsthesia differ in the two cases. If the patient has been taking ether, the circulation will hold out against suspended respiration for a far longer time than if chloroform has been employed.

The colour of the features under ether is usually more florid than under chloroform, and owing to the greater tendency under ether to so-called "holding of the breath," to deglutition movements, and to rigidity of the jaw muscles, temporary interferences with free respiration are more frequent, and hence duskiness is more common than when employing chloroform. Moreover, the intentional air-limitation of ether administration necessarily leads to a minor degree of lividity. It is a mistake, however, to regard moderate duskiness as necessarily dangerous. I would go further than this, and say that in the early stages of ether administration, when employing Clover's inhaler some duskiness is essential if we wish to avoid struggling. A rather bluish colour points to good cardiac action, and if the cyanosis be under control, should not alarm the administrator. Whilst cyanosis is more common under ether than under chloroform, pallor is more liable to arise under chloroform. When chloroform is given in toxic doses, a leaden paleness of the features results; whereas, in ether toxæmia, the colour is blue rather than pale. These dissimilarities in colour are analogous to those in the pulse which have already been described.

The average ether pupil is larger than the average chloroform pupil; but the same causes which produce enlargement or contraction of the one have a similar tendency in the other.

When we come to compare the after-effects of ether with those of chloroform, we must frankly admit that nausea, retching, and vomiting are more common after ether, and that the taste and smell of this agent often persist, to the patient's discomfort, for many hours after the administration.

Ether vomiting is, as a rule, of short duration. It is sharp for the time, but usually subsides before consciousness is regained. Chloroform-sickness, on the other hand, although less frequent, may come on several hours after the anæsthetic has been withdrawn, and is more liable than ether vomiting to prove persistent, troublesome, or even dangerous.

Amongst the after-effects which have been attributed to ether must be mentioned tracheitis, bronchitis, pneumonia, albuminuria and uræmia. These, however, are extremely rare, if, indeed, they ever occur purely from the effects of the anæsthetic. Our knowledge upon the subject is in an unsatisfactory state. Whilst there is an ill-defined but generally accepted belief that ether is more prone than chloroform to leave behind it catarrhal lung affections, very little definite proof exists. Cases presenting such sequelæ after ether have certainly been recorded; but they have also occurred in connection with the use of chloroform. I have administered ether to a healthy adult male for as long a time as three hours and ten minutes, keeping up a deep anæsthesia all the time; and yet no catarrhal effects whatever followed. I have also anæsthetised with ether infants of a few weeks old, persons over eighty years of age, and patients suffering at the time of the operation from nearly every variety of pulmonary affections; but in only three cases have I ever heard of any subsequent complication which seemed likely to have been brought about by ether, and in no single case was it clear that the anæsthetic was wholly at fault. Exposure upon the operating table or immediately afterwards has, no doubt, been responsible in many instances for catarrhal states erroneously placed to the discredit of the anæsthetic.

From the facts which I have placed before you it is clear that we have in ether and chloroform two anæsthetics which are similar to one another in many points, but dissimilar in many others. All carefully compiled statistics point to the fact that

the death-rate under ether is very distinctly less than that under chloroform. Chloroform acts more rapidly and more powerfully than ether; and when once the boundaries of safety have been over-stepped it is more difficult to restore the patient than when a similar accident has occurred under ether. Given that the patient is in a moderately good state of health, it is difficult to place his life in jeopardy under ether; but respiration and circulation may with comparative ease be seriously disorganised by chloroform. If we accept as our highest aim the safety of our patient, we should certainly administer ether, unless this anæsthetic is contra-indicated by the patient's condition or the nature of the operation. As you are all aware, ether is inapplicable in prolonged operations upon the mouth or nose, or when the actual cautery has to be applied to these parts. But in the vast majority of operations ether can be given. Since it has been used more freely in this hospital, fatalities during anæsthesia have conspicuously decreased. With this and other facts before me, I feel justified in urging all of you who are about to enter upon private practice to make yourselves thoroughly acquainted with ether-giving.

THERAPEUTICAL NOTES AND FORMULÆ.

Chlorate of Soda in Cancer of the Stomach.

—At a meeting of the French Association for the Advancement of Science, in August last, Dr. Brissaud, of Paris, made a report upon the above subject. He said that it was an established fact that solutions of Chlorate of Potash exert a very favourable influence on epithelioma of the mouth, and certain forms of canceroid of the face. The successful results obtained with this method induced him to try the effect of the corresponding Salt of Sodium in the treatment of carcinoma of the stomach. This Salt was selected because it is much less tonic and more soluble than Chlorate of Potash, seeing that relatively large doses may be injected into animals without ill effect, and that it is soluble in three times its own weight of water, while the Potassium Salt requires twenty parts of water for complete solution. He had treated several undoubted cases of cancer of the stomach by the administration of Chlorate of Sodium in doses varying from eight to sixteen grammes (two to four drachms) in the twenty-four hours. The results were so satisfactory that if his observations were limited to one or two cases only he would hesitate to publish them, because he might possibly have mistaken cases of chronic gastritis for cancer; for the former, as is well known, sometimes present all the symptoms of malignant disease. But during the last four years he had tried this treatment with equal success in five successive cases of localized gastric cancer, and it is difficult to admit that an error of diagnosis was committed in each of these five cases; this is the more improbable seeing that three of the patients presented a distinct epigastric tumour. In every case the treatment

was followed by the most remarkable improvement, amounting practically to a cure. The drug was given in doses of twelve, fourteen, or even sixteen grammes in the twenty-four hours, and the patients may now be regarded as radically cured. Under the influence of this treatment, the melæna, hæmatemesis, and cachexia disappeared; the appetite returned; and in the three cases in which a tumour could be detected in the epigastrium, it gradually subsided until no trace of it was left at the end of six weeks. On the other hand, the treatment failed in a number of cases, for while Chlorate of Sodium seems to exert a favourable influence on malignant tumours of the epithelial type, it produces no effect whatever on the interstitial sarcomatous type of neoplasms. Moreover, the failure in some of these cases is to be attributed either to the fact that cancer had already spread to other organs, or to the presence of complications which were not amenable to the treatment. For example, in one case the liver was already involved when the administration of Chlorate of Soda was commenced. In another patient, a young woman who had at first experienced some relief from the treatment, the disease suddenly extended to other parts of the body, where it developed with extreme rapidity. She was a patient of Dr. Nélaton; she received sixteen grammes of Chlorate of Soda a day, and in the course of a few weeks hæmatemesis ceased, appetite returned, and she put on sixteen pounds in weight. This improvement was so remarkable as to suggest an error of diagnosis. At the autopsy, however, cancerous nodules were found in various organs and tissues. In a third case death was due to phlebitis of the inferior vena cava. It cannot, therefore, be included in the statistics of the treatment of gastric cancer by Chlorate of Soda.

(Med. Rec.)

Bromoform in Whooping-Cough.—Dr. F. W. Burton-Fanning says that Stepp reported, in 1889, 100 cases of whooping-cough treated with Bromoform without a single failure, and his results were endorsed by other physicians. Dr. Burton-Fanning also reports thirty cases of his own treated with this drug. Bromoform is a colourless, oily liquid with an ethereal odour and sweet taste, insoluble in water. He suspends the Bromoform in a mixture as follows:

Bromoform	℥j
Pulv. Tragacanth Co.	ʒss
Syr. Simpl.	ʒss
Aq.	ad ʒss

For children under one year he gives half a minim, up to three years one minim, up to six years two minims three times a day to begin with, then, if necessary, these doses may with safety be gradually increased until they are doubled. It is important to have a fresh supply once a week, to keep the bottle in the dark, and shake it before taking the contents. Bromoform should never be used when it is of a brown colour, as this is due to its decomposition and the liberation of free Bromide. He considers Bromoform of specific power against the paroxysmal cough, on which the chief dangers of whooping-cough depend. The number of

paroxysms with this treatment are much fewer, shorter, and less violent, and the vomiting always ceases, but the duration of the disease is not materially shortened. If the Bromoform be discontinued within four weeks, the characteristic cough and attendant miseries return at once. He also finds that this remedy is so specific in its action as to be of great use in diagnosis. In doubtful cases its success or failure to relieve the cough led him to a correct conclusion as to the nature of the disease, as subsequently established by other considerations.

(*Boston Medical and Surgical Journal*).

Strophanthus in Pruritus.—Azua has found the Tincture of Strophanthus useful in pruritus due to stasis of the circulation in the papillary layer of the skin as observed in some cases of cardio-pulmonary diseases. He tried in several such cases, and in one of itching caused by jaundice. In the latter it had no effect whatever, but in the others a cure was effected by giving 12 drops twice a day for seven or eight days. He cites the case of a man, aged 70, suffering from emphysema and dilatation of the heart, who had most troublesome pruritus for months. One week's administration of the drug completely relieved the itching. Such being the case, Azua believes that Strophanthus exercises a specific action on the nerve endings.

(Med. Age.)

Neuralgia.—Hypodermic injection of the following solution has been found valuable by Crocq. One cubic centimetre is employed at first and gradually increased to three:—

R. Phosphate of Soda	...	gram. 2
Alcohol	...	" 5
Sterilized Water	...	" 100

Asthma:—

R. Ether	ʒj
Oil of Turpentine...	ʒiv
Benzoic Acid	ʒiv
Balsam of Tolu	ʒij

To be inhaled during an asthmatic attack.

(Med. Rec.)

Non-bitter Quinine:—

R. Quinine Sulph.	...	gr.xv
Acid Sulph. Dil.	...	℥xv
Spt. Menth. Pip.	...	ʒiiss
Sol. Saccharin. Saturat.	...	ʒv
Aquæ Dest.	...	ʒvj

(Medical News.)

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 22, 1893.

A CLINICAL LECTURE ON A BLADDER CASE.

Delivered in connection with the London Post-Graduate
Course at the Sick Asylum, Cleveland Street,
Oct. 26th, 1893, by

THOMAS BRYANT, M.Ch., F.R.C.S.E.* & I.,
Ex-President of the Royal College of Surgeons.

WE have to consider to-day, Gentlemen, such cases as have been provided for us; for, as you are aware, we have to trust to this institution to find material for our lectures. As it so happens to-day we have no great variety to bring before you; indeed, beyond a case which comes before us as a bladder case, and a very obscure one, I have but little to show you. I propose, therefore, to give you a short history of the case as it appears before us, and then to go into the question of its diagnosis, and consider all its different possibilities.

The patient is a man, 61 years of age, who for some years was a potman—until the age of 24; and since then has been a tailor, the latter life probably being a more healthy one than the former. He tells us that he was a fairly healthy man until his present trouble appeared, although he has had a winter cough off and on for the last ten years. He has a rupture in the left inguinal region, which he has had as long as he can remember, and for which he wears a truss. But these points to which I have alluded have really nothing to do with the trouble for which he has been admitted.

We now come to the history of his present illness. A year ago, when he believed himself to be in good health, he had one morning a severe rigor. For this he stayed in bed for the remainder of the day, feeling so ill that he could not go to his work. This was apparently the beginning of his trouble. After a day and night's rest he felt pretty well next morning, and returned to his work. About two weeks later, after he had been at work, and never having previously experienced any difficulty in micturition, he was unable to pass his urine until he went to bed, when, oddly enough, he could micturate in the reclining posture. Since that

period he has had varying difficulty in micturition, passing usually from two to five ounces at a time, there being occasionally sudden and urgent calls to micturate. Micturition was also always easier in the sitting or reclining posture. The urine at this time was apparently perfectly healthy. In this condition he went on for five months, and continued at work. That brings us to about seven or eight months ago, when he first observed that his urine was becoming muddy in appearance and offensive in odour; and these conditions have become gradually more marked up to the present date, that is, up to October 13, when he came in here. He thinks his urine was most offensive about a month ago. For the last five or six months until five weeks ago the patient says he occasionally passed gravel in his urine, ragged pieces sometimes, from the size of a pin's head to that of a split pea; and the passage of these caused him great pain along the urethra; but at no time has there been any blood in the urine. About four or five weeks ago the patient whilst stooping felt great pain in the right side and lower part of the abdomen, and for the first time he observed that there was a swelling in that region. His impression is that this swelling when first discovered was almost as large as it was on admission, when the report states it extended for two or three inches above the pubes. It occupied the lower right half of the hypogastric region to the right of the bladder. The patient thinks it has occasionally become smaller, but he cannot say anything definite about any change in the consistency of the tumour. His pain was increased by stooping, and this compelled him to stop work six weeks ago. He did not, however, consult a medical man until a week before his admission here, when his urine was drawn off for pain and retention. There is no history of bowel trouble—pain on defæcation or constipation—and none of rigors subsequent to the onset of the trouble twelve months ago. He had no abdominal pain except on micturition. Prior to the appearance of the tumour four or five weeks ago, his appetite and digestion have always been good. So much for his past history.

On admission there was a tumour in the right iliac and hypogastric regions. It was hard in parts, with a smooth surface, roundish, and fairly well

defined. No fluctuation could be observed in the tumour, which was tense. It was slightly movable, and handling excited no pain. A prostatic catheter was passed into the bladder, and when drawn forwards after its full introduction, it was made out, according to the report, that the left side of the swelling projected into the bladder; the urine drawn off was quite acid. After the removal of the catheter a few clots of blood came away with the urine, but no pus was seen. Examination per rectum showed no enlargement of the prostate, and no extension of the swelling to within reach of the finger, indicating that whatever swelling there was, and whatever its nature, it did not dip down deeply into the pelvis, or involve the neck of the bladder towards the prostate or rectum. The swelling was evidently high up at the brim of the pelvis. Pus was first seen in the urine in any recognizable quantity on Saturday, the 21st Oct., *i.e.*, eight days after admission. On the following Monday, when I first saw him, the upper part of the swelling was found to be soft and fluctuating.

Having now given you a full history of the case, let us consider what it is; what working diagnosis have we formed? Speaking in general language, it is clearly a bladder case, for most of the symptoms I have related to you are those that are associated with bladder trouble. Indeed, we have now a case in which we cannot doubt there is inflammation of the bladder, cystitis; for we have pus now freely discharging with the urine from the bladder, mixed with mucus in abundance, and the urine is horribly foetid. When cystitis first appeared it would be difficult to say, for the urine was at first acid for a long time, and it is only within the last few days that pus has been seen in the urine, although there had been for some months pain in micturition, difficulty in micturition, and very urgent calls to micturate: all symptoms characteristic of irritation of the bladder, and so we have no doubt that the man has cystitis.

We now go on to ask what connection there is between the cystitis and the tumour. What are the possibilities of the case? Arguing it out from its history we must remember that the man had first a rigor, and it must have been a very severe one, for it kept the man from his work and made him stay in bed. That rigor naturally suggests that at that time there was the beginning of some inflammatory trouble, that is, there was thrombosis of some vessels, giving rise to sudden blocking of

the circulation of a part, and consequently "inflammation." The part involved was clearly some part of the urinary apparatus, for two weeks later there was difficulty of micturition, and relief could only be obtained by lying down. I am not able to explain that odd fact, for I do not understand how it came about. As a rule a man with cystitis or any other trouble about the bladder makes water better in the erect than in the reclining or sitting posture. At any rate, although there was at that time difficulty in micturition, he overcame it naturally, and passed urine which was apparently perfectly healthy. Later on the symptoms of irritability of bladder appeared, with sudden calls to micturate, and still again repeated difficulty in doing so, these symptoms suggesting that, whatever the early trouble might have been, the bladder was becoming more or less involved, and that cystitis had set in. It may be that the whole bladder was not inflamed, but it is enough to suggest that the walls of the bladder were becoming more or less involved in the trouble, whatever that trouble may be.

23-11-93
The symptoms steadily increase in intensity and severity until we have the present marked and characteristic condition of cystitis.

We will now pass on and consider in what way we would explain the presence of this cystitis, and of this swelling? What are the possibilities of the case? Probably one of you might say at once it is a case of tumour, slowly growing, pressing upon the neck of the bladder, interfering with the exit of urine, and so on. Well, that is a theory one must work out. It may possibly be a tumour, and if it is, it is clearly a tumour that has been growing very rapidly indeed. But are the bladder symptoms such as you usually find associated with bladder tumour? I cannot say that I think they are. For a tumour when it involves the inner wall of the bladder, whether its mucous or sub-mucous coat, and penetrates into its interior, is something like a stone or foreign body, and produces at first local irritation, which in its turn sets up painful micturition, frequent micturition, and as a rule hæmaturia; this hæmaturia being mostly a symptom which may be called mechanical, due to the muscular walls of the bladder trying, as it were, to extrude the tumour, by which means blood is squeezed out of it; and this occurs whether the tumour happens to be a fibrous growth simply, a sarcoma, or softer growth, likely to be more squeezable and therefore more likely to be associated with

hæmaturia, or of a kind called papilloma, of a simple or more malignant or infiltrating nature.

With both those varieties of papillomata you get hæmaturia; but oddly enough you do not get *persistent* hæmaturia in all, for in the simple you may get hæmaturia to-day, and to-morrow the urine may be clear and normal, and the patient may go on for a week, a fortnight, or even, as in a case I have seen, for several months, and then another attack of profuse hæmaturia, apparently nothing else but blood and urine coming away. Of course we explain this intermittent hæmaturia with a villous growth in this way. Supposing it occupies the fundus of the bladder or any part of the bladder, not the neck, the little villi are not floated, as it were, into the urethra, and they do not come under the influence of the bladder especially. It is only when the villi are washed, as it were, with a stream of urine into the urethra, and then broken off that you get the hæmorrhage in cases of ordinary papillomata of the bladder. And the same is the case with a villous carcinoma to a degree.

Taking this case as we find it and comparing it with the general symptoms I have suggested to you, as generally typical and characteristic of growth in the bladder, I think the probabilities are very much against much growth being in the bladder itself. It is true I have not yet given you any of the symptoms or results of a local examination of the bladder by the catheter or sound to enable you to come to a diagnosis, but I have not a great deal to tell you there, because I have been particularly careful not to do much with this man, as he is not my patient. Our friend, Mr. Hopkins, who has charge of him here, draws off the urine periodically, and washes out the bladder. I was present on one occasion when that was done, and it was certainly true that the metallic instrument was pressed towards the patient's left side by something in the wall of the bladder, and there was a gritty feel along the right side of the patient's bladder—a gritty feel such as you might find if there was an abraded surface upon which phosphatic concretion was deposited; and with the history that the man has passed small calculi, it is possible that there is a surface upon which phosphatic concretion has been deposited.

Is not that surface then more likely to be the surface of a growth than of an ulceration from inflammation? I quite accept that as a possibility, for if this man has merely an ulceration of his

bladder as the result of an inflammatory action, I think we may be quite sure there would be much more pain than he now has. He is clearly not in such distress as he would have been had his symptoms been due to ulceration of the bladder, the result of inflammation. If there is ulceration it is of a chronic degenerative kind, such as we meet with in cases of degeneration of new growth. So that as regards local symptoms it seems more probable that some growth is encroaching upon the bladder, and has become the seat of some calculous deposit, which is in its turn brought about by the decomposition of the urine, the consequence of inflammation.

If it be not tumour, then, what is it? I wish I could let you examine the abdomen of this patient, but I may not, because it would do the patient no good, but probably harm; so you must accept the report of what I find.

Following out the important rule of looking well at a diseased or injured part before manipulating it, a practice by which a great deal is to be learned, you can see in the case before you that the whole of the lower half of the abdomen is distended, although the chief bulging is in the right lower segment, and that the swelling has a perfectly smooth outline. In passing your hand over the swelling you find it is an ovoid tumour running across the central line up to the umbilicus and extending down into the right iliac fossa. I may say that I think it must be nearly twice the size that it was when I last saw him three days ago. On palpation it is elastic but not fluctuating, and I should say it contains, if not fluid, some remarkably soft solid. In the right iliac region we find the tumour is harder and more solid. Above the tumour there is intestine floating over it. Whatever there is there, is probably situated outside the peritoneum in the connective tissue on the right side of the bladder, showing itself first behind the pubes, and gradually and mechanically so pressing the bladder and soft parts away as to give rise to the present condition in which we find this patient.

When I saw this case three days ago I was much puzzled about it; I am indeed puzzled now, for the case is not clear, but still I think we can make out a working diagnosis. You heard me say that I was sure the tumour was double the size it was last Monday—only three days ago. No growth alone could have increased at such a rapid rate as that; so this fact alone suggests that if growth

exists, it must be associated with some fluid. To explain this rapid increase you may say, May it not be a growth into which hæmorrhage has taken place? Well, that is a possibility. The feel of the swelling suggests the presence of a fluid, but in the absence of fluctuation it is equally probable that we have "a soft solid," a thing of pulpy consistence, such as used to be called "medullary cancers," but which we now know by the name of sarcomatous growths.

When I first saw the case I was led to think that the swelling was fluid, since it was hard and tense, and yet not so hard and tense as to suggest the question of hydatid (because hydatid tumours are comparatively common about the pelvis); for hydatid tumours are remarkably tense so that you very rarely get fluctuation in them. There is also, in large hydatid tumours, a peculiar thrill which you may get through the abdominal wall; but you can rarely get distinct fluctuation. This tumour is globular, but not tense enough for hydatid.

So then we have eliminated the solid growth with hæmorrhage, as a probability, and also, I think, we may say the same of the living hydatid tumour; but I do not think we can *quite* eliminate even the *possibility* of the existence of a dead hydatid associated with suppuration, although I do not regard this possibility as a probable explanation.

Again, may it not be suppuration around the neck of the bladder? May it not be abscess in the wall of the bladder? That is another possibility; and I am somewhat disposed to think that it is a very reasonable probability; for, bear in mind, the trouble began with a rigor; so that we might picture to ourselves a small abscess occurring in connection with the neck of the bladder in front of the connective tissue steadily developing, and then pressing on the neck of the bladder, and so giving rise to the mechanical symptoms of cystitis. It is a probability we must keep before us.

Again, is there a sacculus connected with the bladder? That is another possibility we must not forget. I had such in my mind the other day when a catheter was in the bladder, and the bladder was being washed out; so that I pressed gently with my hand over the swelling, and from what took place I thought, when the contents of the bladder had been pretty well drawn off, that more pus flowed, this fact suggesting that there was a communication between this swelling and the bladder itself, but whether it was a sacculus that had suppurated or an independent abscess I

did not know. The inference was, therefore, drawn that the swelling was not all in the bladder, but had some indirect communication with the cavity of the bladder. Well, it might be asked if it were a sacculus, how is it that the sacculus does not empty itself more readily into the bladder than it appears to do in this case. That is a good argument, for most of the sacculi connected with the bladder have fairly large orifices: either one into which you could put a quill or your finger. But still you do meet with sacculi that are not so, but burrow obliquely through the wall of the bladder, and so have a sort of valvular orifice under which circumstances the communication between the sacculus and the bladder is not so direct as it is in the majority of the cases you see as specimens in the hospital museums.

By way of conclusion, what then is this case? To satisfy ourselves I think we should have to make some exploratory operation, and it is not for *us* to interfere surgically in the case; but by and by I have no doubt the case will be cleared up by what our friend Mr. Hopkins will probably do to this poor fellow in the course of a very short time. But feeling the tumour over again while I have been talking to you and viewing it all round, I certainly come to the conclusion more readily that I did before that it must surely be an inflammatory swelling, mixed with more or less new growth, such as we were led to suspect from the early history of the case. But the true diagnosis we must leave for further investigation and knowledge to decide. As to the treatment of the case I do not think there is a shadow of doubt that the wisest surgical procedure here is to explore and deal with the case according to what is found. If it were my case I should be tempted to explore in the right semilunar line,—not in the middle, because that is where the swelling first appeared.

We have thus then come to a working diagnosis in this case, and have based a line of treatment upon it. How far it is correct, time will prove, as revealed to us by the surgical procedure to which this patient will be subjected.

Note.—Mr. John Hopkins, F.R.C.S., Medical Superintendent of the Central London Sick Asylum, Cleveland Street, writes Nov. 13th:—"A post-mortem examination of the case shows that the tumour grew into a sacculus of the bladder, and hung down over the opening into the bladder, acting as a valve. The sacculus was of great size, and was distended with decomposing urine."

CLINICAL LECTURES ON TWO CASES OF ENTERIC FEVER WITH FATAL ENDING.

Delivered at St. George's Hospital, Oct. 30th, and
Nov. 6th, 1893,

By G. H. WHIPHAM, M.D., F.R.C.P.,
Physician to the Hospital.

I.

GENTLEMEN,—It is not very often that one has two fatal cases of enteric fever within three days of one another, with symptoms—the initial symptoms, at all events—so widely different. I shall take one of them to-day—a very instructive case—and, at the risk of being thought tedious, I shall talk about the other one next Monday, and, if time permit, make a few remarks as to the treatment of the two cases. These two will, when we have done, I think, show how different the initial symptoms may be in cases of enteric fever; that is to say, one may be extremely mild, the other may be very severe, and yet both the patients shall recover in the end, or, as unfortunately happened with those two cases, both of them may die.

The first case was that of a man, aged 22, who came into the hospital on the 4th October. The family history is unimportant.

The personal history was that the patient had had no illness which he could remember. On September 24 he caught cold through going to work without an overcoat, so he says. Three days later he had to give up work, and he has been in bed more or less ever since. The bowels were constipated at first, but he took castor oil occasionally with the result that they became opened three or four times a day. The other, and almost the only prominent symptom was that he slept badly.

On admission he was a well nourished but anæmic man, with a muddy complexion, somewhat heavy eyes, and tongue coated rather thickly but in patches, that is to say, there was a thick coat on the right side, while the left side of the tongue was almost clean, and rather red; the temperature was not high. His pulse was not rapid—80; the heart sounds were clear and distinct.

On looking him over we found the abdomen only slightly distended, so slightly, in fact, that there was a discussion as to whether there was really any distension. There was what my clinical

clerk describes as “a fairly typical typhoid spot” over the iliac spine on the right side. There were many other spots, as indeed one very often finds in enteric fever—acniform spots, erythematous patches (one might almost call them) scattered over the belly, but which are certainly not the typical rose-coloured spots of enteric fever. Careful pressure in the right iliac fossa revealed a gurgling, indicating air and liquid fæces in the bowel. There was no tenderness at all. By percussion the spleen was found to be enlarged; but nothing could be felt of it by palpation. This specimen, which I now show you, is the spleen in question, and is interesting as showing how large the organ may be, and yet nothing be detected of it by palpation. It weighed 14 oz.

The next day the patient was better; that was on the 7th October. The tongue had now become altered. It had lost even the patch of coating on its left, and become raw-looking—devoid of epithelium.

There were light yellow-ochry motions. In fact, there were all the symptoms and signs of a mild attack of enteric fever.

There was every indication of satisfactory progress; two days later new epithelium had reformed on the tongue, and the patient's strength was well maintained. He was alive to his surroundings; he moved his hands briskly and with energy, and equally brisk were the movements of his eyes and eyelids—all three are favourable signs; they showed that the patient was alive to his surroundings, and that there was a reserve of energy left in him.

So things went on till the 16th October with very little change. There was no diarrhoea to speak of; there were some loose motions, but nothing worse. I leave the history here for a minute just to call your attention to one or two of the more important facts I have mentioned.

The history of the onset was really nothing more than the ordinary onset of a fever, that is to say, pains in the back and rigors.

It was a mild case of enteric fever up to the 16th October, that is to say, up to the 25th day of the fever. For a case of enteric fever the temperature was never very high. The prevailing temperature, for the whole 23 days that he was in hospital was never over 101.

Another important point is that there was a complete absence of abdominal tenderness. There were loose motions, but no diarrhoea; the pulse

was pretty strong throughout the whole three weeks; and lastly,—also an important point,—there was, for a case of enteric fever, but little abdominal distension. The whole aspect of the patient indicated a mild attack.

In its clinical aspect there were also these one or two points. First of all, a remarkably slow pulse rate. You know that it is one of the characteristics of enteric fever—this slow pulse with a more or less high temperature. The pulse rate in this case was remarkably slow, as slow as 60 on two successive days.

Then the absence of abdominal tenderness and distension are facts which would seem to indicate the absence of extensive or deep-seated ulceration in the agminated glands. Sir William Gull in a doubtful case of typhoid used to lay great stress on the distension of the abdomen, as being one of the most trustworthy facts in making a diagnosis.

I would call your attention to the condition of the tongue in this case. When the patient came under observation there was a patchy coating of his tongue: the left side was devoid of any coat at all; the right side had a thick white coat, and in the course of a few hours this coat had disappeared, and the tongue was left red and raw. Usually, in the early stages of enteric fever, one finds the tongue which is known practically as the tongue of fever; that is to say, there is generally speaking, a tongue which is sharply pointed when protruded; it is not the flabby indented tongue so often seen in rheumatism; it is remarkable for its red tip and edges, and for the thick white coat in the centre. Later in the disease this central white coat may become thicker and drier, until there is a dark brown or black stripe up the middle of the tongue. That is the common condition in cases of enteric fever; but you may meet with all kinds of variation in its appearance. There is no tongue, I believe, in enteric fever, which is of any special diagnostic value; I can give you an example of this. Some years ago a lad came into the King's Ward under my own care, who had all the signs and symptoms of enteric fever, and yet he had a perfectly clean tongue. It remained clean so long as the fever lasted, up to the time when the oscillations of temperature began, that is to say, up to the time when convalescence began. Then, curiously enough, his tongue became thickly coated with a white fur, and remained so for two or three days. Again the temperature went up and the

patient had a distinct relapse of the fever. As soon as the temperature began to rise the tongue began to lose its coat, and by the time the relapse was fairly established he had a perfectly clean tongue again. At the end of it exactly the same change occurred as before: as the second convalescence became established his tongue again became coated. Now this, of course, is an exceptional case, I mention it merely because I think it may serve to impress upon your memories the great variability of the signs and symptoms of enteric fever.

In fact, there is, perhaps, no disease in which they vary so greatly. Occasionally there is an entire absence of any symptom or sign of the fever; and in such cases it is only by a process of elimination of other diseases that one at last is driven to the diagnosis that one has to deal with a case of enteric fever.

Here, then, ends the first stage of this case; and now I come to the second.

On the 16th of October, the 25th day of the disease, the temperature in the morning was 96.4. The oscillations had begun. That evening the temperature rose to nearly 102. Now we were in some difficulty. It was so far a very mild case, and the patient had been very hungry. I had given him some arrowroot,—a little starchy food in the diet of a fever patient being probably beneficial. I thought that possibly the arrowroot, being undigested, had irritated the surface of the ulcers; and that in this a sufficient explanation of the high temperature would be found. The arrowroot, therefore, was at once discontinued. The temperature came down the next morning to below 99, but that night it went up again to nearly 103. At 103 or thereabouts it remained until the 23rd of October. The pulse rate increased at the same time. There was now no doubt that the patient had a relapse of the enteric fever; and, as the sequel proved, it was a fatal case of relapsing enteric fever. These cases of relapse of enteric fever are common enough; but the fatal cases of relapse of enteric fever are much less common.

There are one or two things that may be noticed as peculiar in this relapse. As I said, the temperature bounded up and remained as high as 103; and for the first day of the relapse the evacuations were ochry. A perfect and complete change seemed to have come over the whole aspect of the case. From a mild case of enteric fever on the 16th October, we were the next day

in the presence of the disease in its most virulent form.

The evacuations of the patient on the 16th Oct. entirely changed their appearance and assumed a very unusual appearance. They contained small white masses of undigested milk, and, what was most peculiar, instead of the usual ochry colour which one sees in enteric fever, and which is more or less characteristic of it, they were of a bright grass-green colour, so that really each evacuation resembled small curds floating in spinach water. I do not recollect ever to have seen such a bright green colour in the fæces of any patient, and certainly not in the case of a patient suffering from enteric fever.

These bright grass-green motions lasted until the eighth day from the commencement of the relapse, and then they became again of the usual ochry colour. I said that these small white masses in the stools were undigested milk; but it has doubtless occurred to many of you that they might possibly have been sloughs from the ulcerated Peyer's patches. We considered that question, and portions of these white masses were submitted to the microscope. They were then found to have no remains of intestinal follicles, and no epithelial or organised substances could be detected in them. That was one peculiarity in this case.

There was another. The relapse was attended by a remarkable alteration in the pulse rate. During the primary attack the pulse rate had only on one occasion reached 88. On all other days it had been below that rate, and on two days it had even been as low as 60, viz., on the 19th and 20th days of the primary attack. On the day before the patient's death the pulse rate was as high as 134; and in the course of 24 hours after the relapse it had reached 120. You will notice the complete change in the aspect of the case. Now a very high temperature, and a very high pulse rate; then a moderately high temperature, and a very low pulse rate. In the first attack the temperature never exceeded 101, in the relapse it was never below it except on one or two occasions quite at the end of the case. The difference in the severity in the two attacks was most remarkable.

A further difference in the primary attack and in the relapse lay in the action of the bowels. In the primary attack the patient says he took castor oil, which caused three or four actions of the bowels a day before his admission. But on no occasion

while he was in hospital up to the date of the relapse were there more than two actions in the twenty-four hours. So that there was practically no diarrhoea at all; there were loose motions, but no diarrhoea. In the second attack the diarrhoea was a prominent symptom, and the motions in the last few days of his life were 7, 5, 5, 4, 4, 6, and 7 on the day of his death. His tongue was the tongue of fever, similar to what I have described as the ordinary tongue of fever. It became red at the tip and edges; then it became thickly coated, and eventually dry, cracked and of a red-brown colour.

In spite of all these bad symptoms it is curious that during neither the first attack nor the second, was there any abdominal tenderness, nor was there much abdominal distension. And that is the more remarkable when one looks at the ulcerated intestines as they are exposed in this tray.

Let me call your attention to another point in the case, viz., that, especially in the last attack, the patient lay on his back in bed with his knees drawn up. Now that is a symptom on which I always lay considerable stress. It is either a good symptom or it is a bad one. As a good symptom one learns from it that the patient is regaining strength and recovering energy. He is well enough and conscious enough to feel that he is uncomfortable in lying for many hours together fully extended on the bed, and so as his strength and energy return, he draws up his legs by way of changing his position. This in a case of enteric fever may be an early sign that the patient is convalescent.

In some cases I have seen the knees drawn up before the temperature or the pulse have become very much affected. On the other hand, this raising of the knees may be a bad symptom, and it is so when the ulceration has extended through the mucous and muscular coats and the inflammation has reached the peritoneum; and this case, I think, shows that the elevation of the knees was a bad symptom probably: the patient, though unconscious of abdominal pain, found himself more comfortable when the pressure of the abdominal walls on the ulcerated intestines was diminished. It is an important feature in any case of enteric fever, and I always watch for it.

In this case, then, one has regard to various facts, some favourable, some very unfavourable. For a bad case of enteric fever to have little or no abdominal distension is certainly unusual; and I think that one was justified in saying at the begin-

ning of this case that in all probability there was not much ulceration of the bowel. While the relapse lasted the majority of the symptoms indicated deep and extensive ulceration.

What one did not like about this man was that he lay, as Sir Thomas Watson so well described it, "down in the bed." This symptom occurred at the last; when once in this position he was too apathetic and weak to make the effort to change it, and he remained thus "down in the bed."

Such was his condition until the 10th day of the relapse, the 35th of the fever; and then one of the motions contained a considerable quantity of red blood. That was the first indication we had that anything had really gone wrong. At 1.30 the next morning there was a profuse hæmorrhage from the bowel, which kept on oozing per anum more or less until 5 o'clock, when he died from syncope.

This is the story, Gentlemen, of one of the most interesting cases of fever I have had to do with for some time; remarkable in its mild onset, remarkable in its severe relapse: remarkable also for the gravity of some of its symptoms, and the comparative mildness of others.

I wish to show you before we part, the condition of the intestine. You see here typical ulcers of enteric fever. You see, first of all, ulcers which are almost healed in one part, and in another deep recent ulcers consequent upon the relapse, but still not deep enough to inflame the peritoneum, and yet, with all this, there was that absence of abdominal tenderness to which I have already alluded.

The ulcers, you will see, have the characteristic adherent bile-stained sloughs.

Notice particularly the remains of the ulceration of the first attack. Immediately above the ilio-cæcal valve all the inflammation and sloughs have disappeared, and the base of the ulcers is quite healthy and evidently healing. If we look higher up the intestine, 9 to 12 ft., we find ulcers in a still more advanced stage of repair: they are almost entirely healed over, and showed, at the time of the autopsy, the delicate vascularity of new tissue. Notice, too, that although one of the ulcers is nearly healed, the intestine is not contracted or puckered in the least.

In next lecture, Gentlemen, I shall bring to your notice the second case in which there was also a fatal result.

II.

THIS afternoon, Gentlemen, I propose to consider with you the second of the cases of enteric fever which I mentioned last week.

The two differed widely in their clinical aspects.

Just a few words, first of all, as to the history. I shall not give it you in detail, but merely in outline—the principal points in the case.

The two cases were alike in this—an unimportant point, but still one of similarity—that there was no family history of any moment. In this case, as long as three weeks before admission, the patient, a fishmonger, æt. 19, felt unwell. He had a cough and general malaise; and, as patients are very apt to do, he attributed his symptoms to getting wet one evening. He also had pain in the back; but he had no other adverse symptoms at all. He went about his business, not feeling well, but for all that was not compelled to lie up. He worked till three days before his admission; that is to say, for probably the first eleven days of his fever this man performed his daily work. On the third day before his admission into hospital profuse diarrhœa came on, and the evacuations were said to be very offensive, but no notice was taken of the colour. On admission the patient's aspect was heavy, stupid, apathetic; his face was flushed, his lips were tremulous and covered with sordes. He had a coated tongue; and a rapid pulse (120), compressible. The abdomen was full when he came under observation; but it moved naturally with respiration. On examining the man's belly it was found to be covered with a very profuse eruption of the typical rose-coloured, lenticular spots of enteric fever. Some of these spots, however, were darker in colour, and these latter did not disappear on pressure. There was a good deal of diarrhœa, and the motions at first were rather darker in colour than the evacuations of enteric fever, and very liquid. There was meteorism; that is to say, the belly was distended, and very tympanitic, even over the splenic area; and (in great contrast to the case of last week) the belly was intensely tender, not only in the right inguinal region—it was more tender there than elsewhere—but also all over. You could not put your hand, however lightly, on the abdomen without the man complaining of pain.

I should tell you that he was a man of extremely nervous temperament; he looked scared and frightened when he came in, and he possibly in

some degree exaggerated the amount of tenderness of which he complained.

Clearly we had to do with a bad case of enteric fever. The temperature ran up on the second day after admission to 104° , where it remained that evening; the next morning it was rather under 104° ; but, with one other exception two days later, the temperature afterwards was below 104° . For all this he took his food well, but without much appetite. So the case went on with very little change, except that on one day he complained of pain and soreness in his back, and he became rather more shaky, with a dry tongue and lips.

From the day of admission, October 10th, quite up to the end of October, the fever continued with nothing very characteristic or calling for any special remark, except that on the 14th of October there was slight hæmorrhage from the bowel; and there was a recurrence of the hæmorrhage on the 19th and 21st. You observe in this case I have not stated the day of the disease. It was impossible to determine the exact day on which it commenced, seeing he had been about his work all the time, and our only note is that it was *about* three weeks before he came in that he began to feel ill. I am obliged to give you, therefore, the day of the month instead of the day of the disease, which, I think, is the better rule because it affords a clear idea of the date of the fever; and knowing this one knows also the condition of the intestine which is all-important.

The eruption lasted to the 21st October. From the 10th to the 21st it was most profuse, and recurred in successive crops. On the 21st it began to fade. On the 23rd (an important day in the case) he became shaky again, but without any increase of distension of the belly, rather less if anything, and also, if anything, he had rather less tenderness. The motions were reported to be partly formed. This was the condition of the patient on the 23rd October. On the same night he was delirious, and in his delirium he sat up in bed and tried to get out of it. In the early morning a sudden change took place. He became covered with profuse perspiration, and complained of intense tenderness and pain all over the belly, which became more and more distended with less and less respiratory movement. The patient, in fact, passed into a state of collapse.

Now recognizing that it was enteric fever we had to deal with, there could be no doubt about what had happened. Perforation had taken place. I

here show you the intestine with its ulcerations in one of which perforation has taken place about $2\frac{1}{2}$ or 3 feet above the ilio-cæcal valve, a very common site of the accident.

The occurrence of perforation of the intestine in enteric fever is almost certainly fatal, and this case was no exception. It is an interesting one; it is, alas! not an uncommon one. It is one of those that are spoken of as "ambulatory typhoid." I think this is anything but a good name. I am quite aware that in more or less poetical language a pestilence is said to stalk through the land; but I do not think one should talk about a fever "walking about." However, I do not think names very much matter so long as you understand exactly their clinical significance, and what is to be anticipated when a patient has been walking about with enteric fever on him. If we set ourselves to correct every misnomer in medical language, our hands would be pretty full. What is meant by "ambulatory typhoid" is that the patient who is the victim of the disease does not rest, does not lie up as he should do from the first. This may be partly because the initial symptoms are so slight that he does not feel that he is really ill, he only feels slightly "out of sorts," he does not perform his duties satisfactorily or comfortably, but still he does not feel really ill; and even if he does many a man, owing to his pluck, or perhaps, more accurately, his foolhardiness, continues to work until he is compelled to give in from sheer exhaustion,—the effect of the poison on the nervous system.

These cases of ambulatory typhoid are among the most serious and the worst we have to deal with. At the same time there can be no doubt that with many of these fevers, not only enteric, but with scarlet fever especially, patients may be out and about the whole time without being conscious that anything is seriously the matter with them. Such an instance has recently occurred in the hospital. I was asked during my last in-week a month ago to see the cook, who had a red eruption on the skin. With an epidemic of scarlet fever in the city, you may be quite sure that all symptoms pointing to that disease were carefully and fully considered. The patient was rigorously examined. Erythema there was, no doubt, but there was no other symptom at all. She did not look ill, and she said she felt well. She had no sore throat, no coating of the tongue, no unusual redness of the tongue, no rise of temperature, and no acceleration of the pulse. She had nothing in the way of symp-

toms at all except a bright erythema of the body, more especially bright on the back, and slight erythema of the extremities.

I may remark in passing that it occasionally happens that one sees such an erythema as this as a prodroma of enteric fever. You know that red rashes are prodromata of other fevers. They occur particularly in smallpox and in enteric fever, and although they are not common, they are by no means rare.

The diagnosis of this case, then, was that it was a case of rather acute erythema; but, as a matter of precaution, she was kept apart from everybody, and the erythema rather rapidly faded; from the back, where it was brightest, last of all. In a day or so, however, a little roughness of the skin of the face was observed, then a roughness of the skin round the neck. Still it was thought it might be simply the desquamation that sometimes follows acute erythema. However, in the course of a few days later there was no doubt about the nature of the illness, as desquamation had commenced in the hard portion of the skin of the palm of the hand, and when I saw her yesterday that desquamation was progressing in the usual way. the skin was peeling off the fingers and palms of the hands, the feet being similarly affected. It was therefore without doubt a case of scarlet fever, with no other symptom except the erythema.

Notwithstanding the fact that many patients with fever do go about their work during the whole of its course, and among them doubtless are cases of enteric fever, the cases of ambulatory enteric fever are usually very severe because the patient has overtaxed his strength. He is at work when he is unfit to work, and he is at the same time trying to withstand the action of a fever-poison, without allowing himself the rest requisite to enable him to do so. The result is that his powers of resistance fail; complications, such as bronchitis, congestion of the lungs, intestinal hæmorrhage, or perforation supervene, and death is the outcome.

It was not long ago that I saw just such a case. The patient had been ill for nearly a fortnight. He was a strong, powerful man, and thinking he was bilious he took some purgative medicines. Amongst other things he took two pills containing Calomel, and they purged him sharply, but did not in any way relieve his symptoms. It was then that I was asked to see him. In addition to the history that he had been ill about ten days, I was told that on

the previous day he had been so far himself that he was proposing to go to Newmarket to see some horses he had in training, but that as he did not feel up to it he had decided to remain at home. His friends also told me he had been so odd in his manner that they had sent him to bed—he had, that is to say, been sitting under the trees on the lawn trying to read a newspaper, and after remaining there about a quarter of an hour he began to talk nonsense, so they thought he had better go to his room. He had not long been there when I saw him. He had high temperature, a rapid pulse, and coated tongue. The belly was somewhat distended but not very much so. It was also tympanitic, and there was gurgling in the right iliac fossa. He was covered with a profuse eruption of enteric fever, and he was very much exhausted. A medical man—a friend of his—took charge of him. Treatment was of no avail, and to make a long story short he was dead in a week.

Many such instances could be quoted, but this one is a sufficiently good example of those cases which are called ambulatory typhoid.

It has already been stated that the eruption in the case we have now under consideration was remarkably profuse. Now the question of the relation between the abundance of the eruption and the severity, or otherwise, of the attacks, is one which has been often raised. My own experience is, that there no such relation exists. I have seen cases of extremely profuse eruption, some of which have recovered without bad symptoms, while others have been rapidly fatal; on the other hand, I have seen cases where there were only one or two spots or perhaps none; and yet, in some of the patients the disease followed a mild course, while in others it was fatal. My own experience, therefore, rather goes to show that there is no definite relation between the amount of the eruption and the severity of the disease.

Let me for a moment direct your attention to another circumstance in the clinical history of these two cases, and of a third case which has just been admitted into Queen's Ward. I think it would be well that you should carefully note it. It was some years ago stated by a German (I think) that herpes labialis is not present in enteric fever. None of these three cases—the first, which was mild in its onset, the second, which was a severe one, and that now in the wards, which is not a mild one—have had herpes labialis; and, so far as I have seen, one is almost justified in the conclusion that

given herpes labialis the patient is not suffering from enteric fever. This, Gentlemen, may be somewhat strong and bold language to use; but I can say this much, that ever since this observation came to my knowledge I have always carefully looked for herpes labialis in the cases of enteric fever which have come under my care, and I have never yet seen it in an uncomplicated case. If this be the universal experience, we have a most valuable fact in the diagnosis. It would enable us at once to distinguish enteric fever, for instance, from pneumonia, in which herpes labialis is so often a symptom.

The exact date of the commencement of this attack was uncertain; and that is a difficulty we often encounter in enteric fever, simply because the early symptoms are often so mild. Probably in this case the fever had run about half its course. The spots were well out when the patient came under our observation; roughly speaking, the spots in enteric fever appear towards the end of the second week, and, judging also by the other symptoms that was about the period of his disease.

It was on the 23rd October that a sudden change came over this patient. The sudden collapse, the failure of pulse, and the cold sweating indicated that perforation had taken place. It is one of the great dangers we have to deal with in enteric fever. It usually occurs about the fourth week; it may occur in the third week; it may occur in the fifth, and even later. You will see at once the importance of recognizing this fact, because it will save you from the mistake of giving more or less solid diet in a disease in which there has been extensive ulceration of the intestine. I hope in my next lecture to say a few words about the treatment of enteric fever and its complications.

I always like to give examples if I can. The following occurred when I was house-physician here. The School in those days was in Kinnerton Street. The School porter there came to me one day to ask me if I would see his son who had been suddenly taken very ill. I went down to Kinnerton Street, and found the patient, a lad of 18 or 19, moribund. His father said he had been suddenly taken with severe pain in the belly while eating a mutton chop for dinner. I went very carefully into the history, and it came out that he had been for some time under a doctor's care for diarrhoea and other symptoms of enteric fever. It had been a mild attack, and the doctor had taken leave of his patient, and saw him only occasionally.

The boy had felt perfectly well for about a week, and went back, perhaps too quickly, to solid food. The collapse indicated what had happened. Evidently perforation of an ulcer had occurred, and in a few hours the boy was dead. Absolutely nothing could be done for him.

I mention these cases, Gentlemen, in order to impress upon you the necessity for extreme caution in ordering solid food for persons who have just gone through an attack of enteric fever. No matter how mild the symptoms have been, you must remember that extensive ulceration may have been present even in the absence of direct evidence of it. Remember, too, the case I related last week where there were hardly any symptoms at all at first; and yet at the post-mortem we found there had been much ulceration of the agminate patches over a considerable extent of the bowel.

A LECTURE

ON

PELVIC HÆMATOMA AND HÆMATOCELE.

Delivered in Charing Cross Hospital, October 26th, 1893,

BY

AMAND ROUTH, M.D., B.S., M.R.C.P.,

Obstetric Physician, with care of Out-Patients, at Charing Cross Hospital; Physician to the Samaritan Free Hospital for Women.

GENTLEMEN,—I have chosen as the subject of to-day's lecture one we have not taken before in the post-graduate course, viz., hæmorrhages into the pelvis not apparent externally, more or less encysted. They come under the two heads of pelvic hæmatocele and pelvic hæmatoma. They are much more common (the milder cases) than is generally supposed.

Roughly, one may divide these hæmorrhages into

- (1) *Those inside a viscus*:—such as, for instance, hæmatocolpos, or hæmorrhage into the vagina, hæmatometra, hæmatosalpinx, and then the more unusual hæmorrhage, hæmorrhage into the ovary (apoplexy of the ovary).

Then there are

(2) *Hæmorrhages into the connective tissue* (hæmatomata). Of these there are two main types:—

(a) Vulvar and vaginal hæmatoma;

(b) Broad ligament hæmatoma.

And then comes the most important of all:

(3) *The intra-peritoneal hæmatocoele.*

The broad ligament hæmatomata are so called because they are not in the peritoneum; they are outside it, beneath it, between the folds of the broad ligament: whereas "hæmatocoele" is the term reserved for hæmorrhage into the peritoneal cavity.

I. INTRA-VISCERAL HÆMORRHAGES. I do not propose to say much about them.

Hæmatocolpos combined with *hæmatometra* is almost always the result of some congenital malformation;—an imperforate hymen, or some atresia vaginae, generally near the orifice, but not necessarily, is the cause of them. Of course this condition is not apparent till menstruation occurs: then a tumour is gradually formed in the vagina and in the uterus, and these organs gradually become more distended, and finally the vulvar orifice bulges, especially at each menstrual period. The treatment is to antiseptically incise the membrane and allow the viscid fluid to escape. We shall not, however, go into that.

Then very often there is what is more difficult to recognize,—

Unilateral hæmatometra or hæmatocolpos, in cases of double vagina where one is patent and the other is not. They form a cystic accumulation of grumous, sometimes gelatinous material on one side of the vagina; and if it is not remembered that the vagina and uterus are made out of the two Muller's ducts united in the centre, longitudinally, it will be difficult to understand what the cases are. What they simulate most is a patent and distended Gartner's duct which would be somewhat in the same position.

Hæmatometra alone is much more commonly the result of some cicatricial contraction at the neck of the womb. I have seen it after the negative pole has been used inside the uterine cavity where Apostoli's treatment has been adopted. It occurs also sometimes spontaneously antecedent to the menopause, due to cervical atrophy.

Hæmatosalpinx very often accompanies these cases of hæmatocolpos and hæmatometra, but occasionally is present without them; and then it is

due generally to tubal menstruation (which some think always occurs), due to the hæmorrhage not having found its way into the uterus owing to some valvular or cicatricial or inflammatory contraction there, and remaining in the tube. Of course, another cause of hæmatosalpinx is tubal gestation, hæmorrhage into a tube having occurred as a result of separation of some of the oval membranes, leading occasionally to the formation of an apoplectic ovum in the tube, or to its rupture.

II. HÆMORRHAGE INTO THE CONNECTIVE TISSUES. Here again I shall be very brief.

(a) *Hæmorrhage into the vulvar connective tissue* forms a hæmatoma; and its chief importance is that it sometimes comes on at an awkward moment in parturition, and may, if it forms a large tumour, definitely obstruct the passage of the head. And practically that is the only case where a vulvar or vaginal hæmatoma really needs interference. In an ordinary case, coming on, for instance, in the course of pregnancy, when the veins are varicose, or in an ordinary congested patient during the act, possibly, of defæcation, a little judicious pressure,—packing the vagina, with external pressure,—will suffice to promote absorption, which will usually occur spontaneously as in cephalhæmatoma of a new-born child's head. But in the cases where a hæmatoma really obstructs labour, it may be necessary sometimes to incise the tumour, let out the clot, and then deliver the child as quickly as possible (with forceps by preference), and to compress or pack, according to its position, the resulting cavity which speedily contracts up, closing by granulation. The difficulty is that one cannot pack the vagina in these cases, and so it is better, if it tends to fill again, to incise and pack, using rigid antiseptics.

(b) *Subperitoneal hæmatoma of the pelvis.* The broad ligament hæmatomata are more common than is generally thought. One sees them pretty frequently, and the important thing is to recognize them, and to know exactly why they should occur.

There seem to be two main sources of the hæmorrhage. The commonest, according to most writers nowadays, is the downward rupture of a tubal gestation, a rupture generally about the middle third of the tube; and if it occurs in the floor of the tube, the hæmorrhage, instead of being into the peritoneal cavity, may be between the layers of the broad ligament,—a very much more advantageous position for the hæmorrhage to be in than the other form, because resistance is offered

by the connective tissue of the broad ligament and by the broad ligament itself, so that the hæmorrhage is generally localized.

The other cause of the hæmorrhage is what is called a parovarian varicocele. There are sets of veins, in the pampiniform plexus mainly, some of which are between the Fallopian tube and the ovary, others are below the ovary, and they are definitely between the layers of the broad ligament. These may, and, according to German authors, very frequently do, rupture, leading to small extravasations of blood; but they may be sufficiently large to cause inconvenience, and even to produce syncope.

They are, as one can naturally see, generally unilateral; but cases are occasionally seen where, apparently, the hæmorrhage has spread along the back of the uterus, stripping off the peritoneum, and getting round to the other side. One questions whether they are pure cases of hæmatoma, or whether they are not partly inflammatory; but still they are said to be purely hæmorrhagic, and, I suppose, if the hæmorrhage was very severe, and the vessel ruptured was a large one, this result might follow.

The symptoms due to broad ligament hæmatoma are, first of all, that there is generally the history of some strain. In the case of the tubal gestation this is not necessarily the case; however, the rupture taking place spontaneously owing to continued growth and thinning of the foetal coverings. But, at all events, there is a definite statement of shock, not coming on instantaneously, as in some forms of intra-peritoneal hæmorrhage, but of more gradual onset, although fairly sudden as compared with anything like an inflammatory attack. Then, following that pretty rapidly, comes on pain, due to the separation and stripping off of the coverings of the broad ligament. With that follows pressure—pressure on the uterus, pressure on the bladder generally, pressure to some extent on the ureter (if the base of the broad ligament is involved, as it sometimes is), and pressure on the vessels in the pelvis, and a certain amount of reflex irritation of the bladder always accompanies it, so that there may be retention or frequency of micturition. If the hæmorrhage is at all extensive the anæmia is well marked, and, in the first day or so, at all events, there is no pyrexia; and this latter point is very important to remember, as opposed to a broad ligament phlegmon, where a rigor and pyrexia occur shortly after pelvic pain.

Per vaginam, one finds on one side, where the broad ligament is known to lie, a distinct tumour, generally a rounded tumour in the first instance, not very hard but rapidly becoming so, and it dislocates the uterus completely over to the other side, and, at the same time, usually fixes it. The fixation of the uterus depends rather upon the position of the hæmorrhage. For instance, if the hæmorrhage is in the middle portion of the broad ligament, not quite up against the uterus, naturally it does not quite fix it; the uterus is a little able to move, although it is pushed bodily over. If a tubal gestation, for instance, were to rupture in its middle third, there would not necessarily be a sufficient amount of hæmorrhage to reach the uterus, and the uterus would not therefore be perfectly fixed. At the same time one feels by the examining finger that Douglas's pouch is empty. At the first stage there would certainly be no exudation there, and the fact that Douglas's pouch was empty, with these symptoms of internal hæmorrhage, would show that it was not an intra-peritoneal case.

And that is the first thing that one has to make out—the position of the hæmorrhage. It is easy to assume that hæmorrhage is occurring somewhere, the difficulty is to make out whether it is intra-peritoneal or extra-peritoneal. From the points I have laid down the diagnosis is sufficiently obvious. The connective tissue is almost entirely on the sides. There is a little in front, and a little in the folds of Douglas underneath the peritoneum; but not sufficient to make it other than fairly true that the connective tissue is mostly on the sides of the pelvis, whereas the peritoneal spaces are mostly behind, except of course, the utero-vesical pouch, where, fortunately, these hæmorrhages hardly ever occur.

Another point of diagnosis, as I have partly said, is to make sure that we have not to deal with a case of parametritis, inflammation of the cellular tissue. And there the onset helps, being very sudden in hæmatoma. There are symptoms of hæmorrhage, anæmia, sometimes even to syncope; and, in addition to the fact that pain is not a very prominent symptom, there is no rise of temperature at first, very often none at all from beginning to end; in fact, there may be an abnormally low temperature. The position of the parametric exudation, like a hæmatoma, is not always quite up against the uterus. The common cause of a parametric exudation is septic absorption; and the most typical form, is where the cervix has been fissured on one side in

the course of a labour, and absorption goes on into the cellular tissue of the broad ligament, and fixes the uterus by the exudation being absolutely continuous with the uterus. In the later stages there is sometimes a gap between the phlegmon in the broad ligament and the uterus, because absorption has gone on where it has originally started, and the exudation has travelled sometimes right away to the pelvic end of the broad ligament, or even along remote planes of connective tissue, or along vessels, producing a condition which Matthews Duncan used to call remote parametric phlegmon.

The prognosis in these cases of broad ligament hæmatoma is generally excellent. The hæmorrhage is confined by the broad ligament, and it generally ceases. There is sufficient resistance in the tissues to check the hæmorrhage, so that absorption is the usual finale in these cases. Just leave them quietly alone, keeping the patient in bed and the bowels open, and the patient recovers nicely. If absorption, however, does not occur, it is generally for one of two reasons. Either the effused blood undergoes a change and suppurates. That is generally the result of repeated examinations or attempts at aspiration, or, apparently, sometimes from contiguity of bowel, which, getting adherent to the broad ligament, allows of the passage of gases through the membranes, so making the blood septic. It is, however, comparatively rare for suppuration to occur, and if it does, perforation into some of the surrounding organs generally takes place, generally into the vagina, sometimes into the bladder, sometimes into the rectum, especially on the left side, or more rarely a serious complication into the peritoneal cavity. Occasionally it will strip off the peritoneum in front of the broad ligament and point somewhere below Poupart's ligament. Suppuration then is one of the causes of non-absorption. Another is that the hæmatoma may be due to the rupture of a tubal gestation, the foetus escaping through the rent in the tube into the folds of the broad ligament in the midst of its hæmorrhage. If the foetus remains in organic union with the placenta by means of its cord, and the placenta remains sufficiently attached, the foetus may go on growing. There are many instances of this, the foetus going on growing after primary rupture into the broad ligament, stripping the peritoneum of the broad ligament right up to the front of the abdomen, much as a distended bladder may strip the peritoneum off. The foetus may go on growing

to the seventh or eighth month, or even to the full term, when a secondary rupture may result, the hæmorrhage being then intra-peritoneal, and the patient may die. But the generality of cases are diagnosed before the full term is reached; and at, or about the viability of the child an operation is performed, when it will be found that an incision over the tumour will not have to pass through the peritoneum at all, the foetus being found immediately beneath the integuments loosely encysted and can be removed without difficulty, and often the placenta also. The sac is then drained in the usual way. The treatment, therefore, of these cases in the first instance is, practically, to leave them alone. I have never yet seen a case of broad ligament hæmatoma where it was necessary to interfere unless some suppuration had occurred; and then, of course, it has to be opened like any other abscess, in that position.

III. PELVIC HÆMATOCELE. (Intra-peritoneal.) This is the most important part of our subject. Such cases are probably numerous. A few years ago when Tait made his great discovery of the greater or less frequency of tubal gestation, he believed that, practically, every case of intra-peritoneal hæmatocele was due to the rupture of a tubal gestation; and he also then believed that of these intra-peritoneal cases, 99 per cent. were fatal if untreated. Subsequent experience has proved that both of these statements are incorrect; and it is now known that hæmorrhage may come from a variety of causes, and that, no matter what the cause is, the patients frequently recover. They recover for the reason that the effused blood becomes *encysted*. That makes the whole difference between fatality and the recovery of the patient. If there is no pre-existing or coincident lymph exudation, the patient will certainly die. The blood becomes diffused all over the abdomen, and, short of operation, the patient is very likely to die, whereas if, as often is the case, there has been some pre-existing inflammation, or the hæmorrhage is slow and gives full time for lymph to be thrown out and mat the intestines together, so forming a roof over the Douglas's pouch, the case recovers.

ETIOLOGY.

There are many causes of intra-peritoneal hæmatocele. They may be grouped according to the organ at fault.

(1) *The Uterus.* There are cases of *regurgitation of blood during menstruation*, due presumably to

some form of obstruction, which is not always discoverable. Then there is *rupture of the uterus*. There are two forms of rupture of the uterus. There is the spontaneous rupture, as it is called, which occurs apart from labour, and which, I think, must be put down in almost every case to interstitial gestation (Lewers), gestation in that part of the Fallopian tube which is in the wall of the uterus. There is also rupture of the uterus in the second stage of labour. That we cannot now discuss.

(2) *The Tubes. Rupture of a hæmatosalpinx and rupture of tubal gestation and tubal abortion* practically include all that we have to say about them.

(3) *The Ovaries*. I mentioned hæmorrhage into the ovary when speaking of hæmorrhage into a viscus; and occasionally that does occur; but it is more a curiosity than anything else. The hæmorrhage in such a case is into the Graafian follicles, there being sometimes one large hæmorrhagic follicle with a number of small ones, or the whole ovary is made up of dilated follicles all of which contain blood, in which case *apoplexy of the ovary* is the name sometimes given to it.

On the rupture of a Graafian follicle after the maturation, and during the escape of the ovum into the Fallopian tube, sometimes an escape of blood takes place with it, which may be profuse enough to produce hæmatocele (Winckel).

Rupture of an ovarian cyst must also be mentioned though it is hardly pelvic; it is generally more intra-abdominal than intra-pelvic. An ovarian cyst does not often rupture except from violence; but it may rupture secondarily to torsion of the pedicle, followed by hæmorrhage into the cyst.

Again, there are such things as *varicose veins over the ovary,—ovarian phlebectases*, as they are called (Zwicke). I have never seen a case.

(4) *The Broad Ligament*. I have mentioned the *parovarian varicoceles*. There are also *varicoceles* definitely formed in cases of *old-standing incarcerated retroversion of the uterus*, where the uterus is prolapsed as well as retroverted, and sags down, getting caught between the folds of Douglas. The pampiniform plexus gets nipped on each side, and the veins necessarily dilate, etc.

I have already discussed another broad ligament cause, viz., secondary rupture of a broad ligament gestation.

(5) *The Peritoneum*. Another cause, much

more common than is generally thought, is *separation of peritoneal vascular adhesions*. There is no inflammatory attack in the pelvis without these bands of lymph being formed, sometimes at first merely glueing viscera together, and then in the course of movement of the bowels, or the growth of the uterus in pregnancy or merely due to habitual change in the position or to the respiratory movements of the patient, the adhesions get lengthened out, and being more or less vascular, may bleed very freely if then torn across. In abdominal operations they have often to be tied in two places and cut between the ligatures. In the process of reduction of the uterus in retroversion, or trying to push an incarcerated tumour out of the pelvis, if there happen to be adhesions they are sometimes torn across, and rather severe hæmorrhage may result. These are necessarily entirely intra-peritoneal.

One must also mention *gravitation of blood into the pelvis* from rupture of aneurysms or of abdominal viscera higher up.

There is thus a fairly large group of causes of intra-peritoneal hæmorrhage. Hæmatoceles due to rupture of adhesions are usually not very profuse, but deaths have occurred from them. Hæmorrhage from ruptured Graafian follicles is clearly not an accident of great importance. But cases are recorded where, especially in so-called "bleeders," the hæmorrhage is sufficiently marked every month as to produce such an amount of shock and vaginal physical signs as will enable the diagnosis to be made. Naturally most frequently they will be non-symptomatic.

Rupture of varicoceles, of tubal gestation, and of hæmatosalpinx are sometimes undoubtedly fatal; and those are the most serious of all. Regurgitation of blood from a menstruating uterus is not as a rule fatal from the hæmorrhage; but there are a considerable number of cases where a fatal result has ensued from the blood being more or less septic: and precisely the same result has very frequently happened where an operation has been performed for the relief of a hæmatocolpos or hæmatometra where antiseptic treatment has not been sufficiently rigidly adopted, and the blood has escaped from the accompanying hæmatosalpinx, or from the uterus along the tube, and owing to the vaginal secretions being septic, or becoming so, septic peritonitis has ensued. To prevent this regurgitation, no attempt should be made to evacuate the blood too rapidly. The blood is very viscid

and glutinous, and comes away with even more difficulty than treacle would, and I have seen operators after opening the vaginal membrane press down the uterine tumour from above the pubes, grasping it after Crèdè's method, with the result that the fluid in utero, unable to escape quickly along the vagina, regurgitates through the Fallopian tubes, which may already be in a condition of hæmatosalpinx. The fluid ought to be allowed to take its own time.

(To be concluded.)

THERAPEUTICAL NOTES.

Guaiacol in Typhoid Fever.—Dr. B. M. Baker, writing of the use of Guaiacol in typhoid fever, says:—

"Some three months ago, I had in charge a man thirty-five years of age, suffering from a mild attack of typhoid, who, after having had no abnormal temperature on the twenty-first and twenty-second days, suddenly had the temperature go up to 100° F. at five p.m. on the twenty-third day. On questioning him, I found that ten years previous to that time he had suffered from what he termed bronchial hæmorrhages, and although I found no signs of either recent or old chest trouble on physical examination, yet there was slight cough and scanty mucous expectoration, and I therefore thought it best to put him on Guaiacol in five-drop doses, three times a day. After forty-eight hours the temperature fell to normal, but the cough and expectoration continued. This rapid effect of the drug upon the temperature made me think at once of the possibility of its having the power of arresting the growth of the typhoid bacilli in the intestine.

Since then I have treated seven cases of typhoid with it, and my friend, Dr. C. F. Newbill, at my suggestion, has tested its efficacy in twelve other cases, making in all nineteen cases.

Most of the cases have been mild types of the disease, with few complications and no violent symptoms, and they have varied greatly in ages, from a baby of eighteen months to an old lady of sixty-five years.

We have found no need of antipyretic measures of any sort, and the temperature is kept constantly low, simply by the administration of this drug. With it the temperature falls, the pulse grows less rapid, the tympanites and diarrhoea disappear, the tongue becomes moist and clean, the nervous symptoms rapidly subside, and the patient goes on to a complete recovery. This has been proven in each of the nineteen cases treated by us.

One case, which illustrates more clearly the good effect of the drug, I will mention in brief. I was called to see Martha J.—, a negro girl sixteen years of age, on the night of the sixteenth day of the disease, when I found her with a temperature of 105° F., pulse 135, dry brown tongue, teeth covered with sordes, wildly delirious, picking at the bed-clothes, etc. She had to be kept in bed by force. The abdomen was markedly tympanitic, and she was having frequent pea-soup evacuations. I ordered Bismuth, Brandy, and Guaiacol in five-drop doses, given in whiskey, largely diluted with water, three times a day, after eating; milk and beef-tea made up her diet. The next day the temperature had fallen two degrees, pulse less frequent and stronger, delirium less

pronounced, and diarrhoea and tympanites much improved. Seven days from this time, I found the temperature normal, pulse 80, and patient convalescent. She is now perfectly well.—(*Med. Rec.*)

The Iodine Treatment of Goitre.—Dr. E. Nazaries uses Iodine in the treatment of goitre as follows:—He dissolves from 75 to 90 grains of Potassium Iodide and from 20 to 30 drops of Tincture of Iodine in about 5 ounces of distilled water. A spoonful (tea or table, not stated) of this is diluted with a pint of water, and this amount taken daily during and after meals. Its use must be continuous.—(*Bul. de la Soc. de Pharm. de Bordeaux.*—*N. Y. Med. Jour.*)

Sodium Bicarbonate in Large Doses.—In the *Revue Générale de Clinique et de Thérapeutique* is given some observations on what Huchard calls heroic alkaline treatment. This consists in the administration of two drachms to ten drachms daily of Sodium Bicarbonate, in successive doses, in conditions of hyperacidity of the stomach sometimes present in diabetes, in the gastric crises of tabes, in cardiac disease with acidity of the stomach, in diseases of the liver, and sometimes when gall-stones have formed. A case is recorded of incipient diabetic coma warded off by ten drachms daily of Bicarbonate of Soda.

Opium and Bromide for Epilepsy.—Opium associated with Bromide is advocated by Flechsig in the *Neurologisches Centralblatt* as superior to Bromide alone. Pills of powdered Opium are given two or three times a day, the strength of the dose being gradually increased during a period of six weeks, when the drug is abruptly dropped and replaced by large doses of Bromide of Potassium. The large doses of Potassium are continued for two months. Then they are gradually reduced to a daily portion of 3ss. The Opium has a preparatory effect, facilitating and strengthening the action of the Bromide. During the Opium period of treatment the only amelioration is a temporary suspension of the attacks, or a decrease in their frequency. Two cases are recorded of complete cure by this method, when all other known remedies had failed.

(*N. Y. Med. Rec.*)

ERRATUM.—On page 34 of Vol. III., line 19 from the bottom of column 1, the word "detached" should have been "diseased."

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 29, 1893.

A CLINICAL LECTURE

ON

CHRONIC GRANULAR NEPHRITIS, AND ITS ASSOCIATION WITH PLUMBISM AND GOUT.

Delivered at the School of Medicine, Sheffield, Oct. 31, 1893,

BY

ARTHUR J. HALL, B.A., M.B. Cantab.,

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GENTLEMEN,—You have frequently seen in the wards and in the out-patient rooms, cases of the kind about which it is my privilege to speak to-day. I refer to cases of chronic granular nephritis; and it may have struck you also what a number of these, and often the most marked cases, occur in those who follow the trade of file-cutters. I have therefore put together a few of the cases which have come under my notice during the past year, and wish, in the short time at my disposal to-day, to point out to you some of the more important clinical features of this destructive disease.

File-cutters, as you know, are much exposed to a chronic poison, namely lead, and often come under our observation suffering from plumbism; they also frequently are subject to gout, and this is the more marked because amongst the poorer classes here, typical gout is comparatively rare. Now, gouty people are exceedingly likely to suffer from chronic granular nephritis, so much so, that the disease is sometimes called "gouty kidneys." What the connection between the three is may be differently interpreted, but one thing is certain, namely, that file-cutters do often have chronic granular nephritis, without ever having suffered from gout in any form.

Evidently, then, we may assume on good grounds what your text-books will tell you, that gout and lead may separately cause chronic Bright's disease, and that the two causes are often combined in the same individual. A third cause is given by some as alcohol, by others it is considered doubtful; it seems to me that if alcohol

alone does not produce the disease, it at any rate must act as a powerful aid when the other causes are present. But I venture to think that when we speak of the nephritis as a disease, we are committing an error. The condition of the kidneys is only part of a general disease of, principally, the vascular system; and in the class of cases of which I am speaking to-day, this disease appears to be an arterio-capillary fibrosis.

I will now read you short notes of two very typical cases which came under my notice a short time ago when they were very advanced, both of which died within a day or two of one another.

Case 1. Chronic Granular Nephritis in a File-cutter with Gout.—W.J., *æt.* 41, was admitted into the Public Hospital, under my care, on November 12th, 1892. File-cutter 29 years, married. No history of syphilis. Has drunk beer freely. Had first attack of gout in right big toe 16 years ago.

Family History.—Father file-cutter, had gout. Four brothers, all had gout.

Present Illness.—Began three years ago with dizziness and loss of power in right arm and leg; this almost completely recovered, but was followed by gradual weakness and tremors of the right arm and leg, which prevented him working, and have gradually got worse. Has had much headache lately, shortness of breath, cramp in the legs, and constipation. No colic. Had to get up frequently in the night to micturate during the last two or three years.

Present Condition.—Complexion muddy; temporal arteries tortuous and thickened; topi in both ears; gums pale, blue line; radial arteries much thickened, feel like pieces of solid india-rubber about diameter of a thin slate-pencil; pulse is incompressible at the beat, and almost so between the beats; brachial arteries at elbow very thick and prominent.

Præcordial bulging; apex beat 5th space outside N.L.; cardiac dulness up to 4th rib, not across the sternum; impulse heaving, epigastric pulsation, both sounds loud and thumping.

Respirations frequent; good resonance over chest; breath sounds rough; rhonchi over both bases. Liver edge felt below ribs. Spleen not enlarged. Urine, clear, pale, sp. gr. (?), loaded with albumen, heavy sediment of hyaline and granular casts. No oedema.

Ophth. exam.—Right eye: choked disc, well marked white patches radiating from the yellow spot.

Left eye: disc natural, similar white patches.

The patient remained in the hospital until December 21st, 1892, and improved considerably under diet and treatment. Thus, his albumen diminished, his pulse tension became less, and he felt much stronger. In March, 1893, however, I went to see him at his own home with the assistant house-surgeon (Dr. Knox), and found that he had been in bed some time with extreme prostration, and was having "fits" at short intervals. Pulse, high tension; urine, loaded with albumen; skin very dry and shrivelled; no headache. In the fits he is dizzy, and reaches out for someone to hold him. Treatment proved of no avail and he died March 19th, 1893.

Case 2. Chronic Granular Nephritis in File-cutter with Gout.—T. W. came under my care as an out-patient at the Public Hospital on February 28th, 1893, æt. 46. File-cutter 37 years. Had influenza two years ago. Gout in big toe, typical attack one year ago, for first time. Since then two other attacks. Had some fits nine weeks ago.

Family History.—No gout that he knows of. Brother, a file-cutter, has had rheumatic fever.

Present Illness.—Began three weeks ago with attack of gout in the foot. Since then his breathing has been bad, and he has suffered from pain in the left side on taking exercise.

Present Condition.—Very sallow; anæmic; no tophi; slight arcus senilis; blue line on gums; teeth flat-topped; pulse, high tension, arteries thickened; chest, emphysematous; a few râles at the bases behind; heart, hypertrophy of left ventricle; no murmur, second aortic sound accentuated; urine, loaded with albumen.

Ophthalm. Exam.—Both discs swollen. No hæmorrhages or white patches. The patient got gradually worse, and was visited at his own home by the assistant house-surgeon (Dr. Knox), with whom I saw him just before his death on March 19th, 1893. He has been in bed with much severe headache, wandering delirium, difficulty of breathing, and fits at frequent intervals, in which he loses consciousness, bites his tongue, twitches his limbs, and finally falls off in a dose. The skin is dry, tongue coated. Pulse small volume, high tension, frequent; no appetite; no dulness in chest; few rhonchi; no œdema; urine loaded with albumen.

Putting together the histories of these two cases, there are many points common to both, whilst there are others on which they differ widely. The age at which they died is worth noting, 41 and 46 years respectively. That is, in the prime of manhood, when their capacity for doing work should have been still great; moreover, notice that in *case 2*, who lived to 46, there is no history of drink—in fact, he was a steady man; whereas, in *case 1*, who died 5 years younger, there was a history of excessive drinking. Also in this connection I would have you remark that *case 1*, who inherited a strong gouty tendency, had gout for 16 years; whilst *case 2* had only suffered from gout 1 year before his death. Evidently *case 1*, with his excessive drinking and gouty inheritance, was as good a subject for lead to act on as could be found; whilst *case 2* had neither inherited gout nor taken excess of drink. There is no history of attacks of plumbism in either of these cases, I mean acute attacks of colic, headache, constipation, or affections of the peripheral nerves; and we may presume that such therefore never occurred, as the men themselves know what they are liable to, and soon recognize anything of the kind. I do not attach much importance to this, as in other similar cases I have got a history of colic, etc., which has usually occurred in their younger days, and passed off; one might suppose that the colic

acting as a warning of danger to them, they would take care by extreme cleanliness and other means to get as little of the poison as possible; but my experience is that it produces very rarely a habit of precaution.

In *case 1* there was a slight stroke 3 years before admission, producing right hemiplegia. This, as you know, is a very common occurrence in chronic nephritis, and often the cause of death.

What are the signs and symptoms by which you can recognize the disease under discussion. 1st. *The vascular system.* The arteries are thickened; this you can ascertain by compressing the radial arteries at the wrists, and feeling that they roll under your fingers like pieces of thick string, you can also usually see the temporal arteries prominent and twisted. Again, the pulse tension is high, that is to say, that whereas in a normal pulse it is quite easy to compress it with your fingers in the intervals between the beats, in these cases it requires much greater pressure to do so. And I would strongly advise you whenever you feel a pulse to try compressing it at the beat, and between the beats as a matter of routine, you will then soon learn to notice the difference between a high and low tension pulse. Another sign of the disease of the vascular system is *hypertrophy of the left ventricle*, evidenced by a displacement downwards and outwards of the apex beat, with a heaving impulse. Sometimes the presence of emphysema of the lungs renders the impulse impalpable, as in *case 2*. With the hypertrophy of the left ventricle there is a thumping first sound on auscultation, and a loud accentuated second sound at the base of the heart over the aortic area; this latter being merely an expression of the high arterial blood pressure. Another evidence of vascular degeneration is to be found in the hæmorrhages which readily occur in various parts.

2nd. *Changes in the urine.* Its quantity varies, it is usually increased in the earlier stages, but diminished towards the end. Thus, in *case 1*, the average amount passed in twenty-four hours during thirty-nine consecutive days was only 38.4 fl. oz., instead of the normal 52 fl. oz. There is usually found to be nocturnal micturition, that is, the patient has to get up once or twice in the night to urinate. The sp. gr. of the urine is low when the quantity discharged is great, but gets higher as the quantity passed diminishes. *Albumen is present* sometimes in fairly large quantities as in both the above cases, at other times merely

a trace, which may easily be overlooked from two causes, firstly, because the test is not performed sufficiently delicately and carefully; secondly, because the sample of urine is probably passed just before examination, instead of being a sample from the mixture of all the urine passed by the patient during twenty-four hours.

As regards the first of these two causes of fallacy, it is best obviated by using, whenever possible, the cold Nitric Acid test, and waiting for a few minutes before deciding whether albumen is present or not; moreover, if the case is one of considerable doubt, confirming the result by one or more other tests for albumen, such as boiling, —or adding Acetic Acid and Potassium Ferrocyanide by the simple and portable urine-testing tablets of Dr. Pavy. *Casts are usually present*; in case 1 they were hyaline and granular, and very numerous. As regards looking for casts under the microscope, I find most students, even advanced ones, have great difficulty in knowing what sort of an object they are to look for, a fact I venture to think greatly due to their having often only seen kidney tissue from the point of view of microscopic sections. If, the method described in Prof. Stirling's excellent treatise on Practical Histology,* and which I have used with great value in the practical course here for the last two years, be adopted, you will see the urinary tubules disintegrated, and by very simple means will get a far more vivid idea of the kidney structure than from sections alone. You will then be much aided in your early searches for tube-casts, for they are merely moulds of the urinary tubules.

3rdly. *Changes in the Fundus Oculi*.—In case 1 these were very typical, there was swelling of the optic discs, so that the vessels could not be seen in continuity, and the outline of the disc was blurred. Besides this there were very typical small white patches radiating from the yellow spot.

In case 2 the discs were swollen, but there were no white patches.

In conclusion, I would draw your attention to the very great value you will derive from a steady perseverance in using the ophthalmoscope. It will often come to your assistance in those very cases where aids to diagnosis are extremely scanty, and a correct diagnosis of immediate importance.

* Take small pieces of kidney and place them in HCl for four hours. Place in water tinted with Iodine for twelve hours. Mount in Farrant, and press down coverslip.

A CLINICAL LECTURE ON THE CLINICAL IMPORTANCE OF "EAR-ACHE" AND "RUNNING FROM THE EAR."

Delivered at the Middlesex Hospital, Oct. 31, 1893,
By HENRY MORRIS, M.A., M.B., F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—I am going to take advantage of a case of abscess in the mastoid cells, which has recently been under my care in Broderip Ward, to make some remarks to you on certain affections of the middle ear, which are very likely to be overlooked or misunderstood by you when you meet with them in practice, unless your attention has been drawn to them during your studies here.

I will first read to you the history and description of the case, and you will then be better able to appreciate the lessons which it teaches.

The patient, H. T., æt. 57, is a man of medium height, well-built and muscular. He says he has always enjoyed good health; to use his own words, he has "never been ill a day in his life." His present illness began after he had been working for three or four weeks in a draughty shop. He had previously had no sore throat, and no pain in the ear. The first thing he noticed that was wrong was a neuralgic pain in the occipital region. After this had existed for two or three days he was aroused one night whilst lying down by hearing a sort of "explosion" in his right ear, and found that his ear was damp,—that discharge was coming from it. This happened on the night of the 21st September. On the 23rd he went to the Ear and Throat Hospital, where they told him he had an abscess in the drum of his ear which had broken.

Now, it is particularly noteworthy that the man says that at first this discharge was of a red colour, but that it afterwards became paler, and then "like matter," *i.e.*, purulent. After the first onset of discharge from the ear he lost all his pain in the occipital region; and, in fact, from that time till a few days before his admission here, he had no pain whatever. He kept a plug of cotton wool in his ear, which, he says, was always soaked with discharge, and when he removed the plug there was a running discharge. This discharge from his right ear went on (he having no pain, no deaf-

ness, no sore throat, no trouble of any kind) for a month and three or four days. At the end of this time he began to have pain, redness and throbbing sensation over the mastoid region of the same side and down his neck for 2 or 2½ inches.

In this condition he applied again at the Throat Hospital, where it was explained to him that the swelling was of a serious nature; and that his was not a fit case for that hospital. Being advised to go to a general hospital, he came here.

On admission we found that there was a hard, red, œdematous, and tender swelling of oblong outline, over the mastoid process, and extending down the side of the neck for about 2½ in. The swelling down the neck did not take definitely the line of the internal jugular vein, and it spread laterally over a much wider area than the vein. The skin pitted very easily on pressure. From his ear there was a considerable amount of purulent discharge. His admission with this state of things took place on October 19. The following afternoon I saw him, and he was then pretty much in the same condition as above described. It was very clear that the month of suppuration from the internal ear had led to a suppuration in the mastoid antrum, and that, in order to relieve him and prevent the serious consequences which follow mastoid abscess he must be trephined. This I did at once.

The operation consisted of the following steps. The patient having been anesthetized, an incision an inch and a half in length was made directly down on the mastoid bone dividing the periosteum. There was very free bleeding from a number of small vessels in the inflamed integuments, which was controlled by pressure forceps. A small quantity of pus escaped as soon as the periosteum was divided. The periosteum was raised, and the mastoid process was then carefully examined, and, just behind and on a level with the ear-hole, a small opening, large enough to admit a probe, was discovered. Through this the probe was passed inwards and forwards. A trephine a quarter of an inch in diameter was then applied over the part of the bone where this opening was; the point of the trephine indeed fitting into the opening through which the probe had been passed. In this way a small circle of bone was removed, and at once there escaped a drachm or two of pus from the mastoid antrum, which was enlarged owing to the destruction of the partitions between the

mastoid cells. A probe was passed into the antrum, and another along the auditory canal, and it was thought the probes impinged upon each other, but some doubt was expressed about that. There was no doubt, however, that the posterior wall of the tympanum was rough and bare of its lining membrane. The next step was to clean out the cavity of the mastoid antrum, and this was done by putting little pieces of cotton wool upon the end of torsion forceps, and rubbing and swabbing the walls of the cavity. After this was done, in order to make sure the cavity was thoroughly cleansed it was well irrigated by a 1-2000 solution of Perchloride of Mercury. The auditory canal was also thoroughly irrigated in the same fashion. A drainage tube was then put into the antrum. The lower portion of the wound was closed by sutures; the parts about the ear were covered with antiseptic dressing, in which was cut an opening for the auricle. After the operation was over the man returned to bed, and from that time he expressed himself as feeling decidedly better. All the pain in the mastoid region and down the side of the neck at once subsided. The redness and swelling about the neck rapidly subsided. The amount of discharge, too, has gradually decreased, until now from the opening behind there is scarcely any, and from the ear very much less. The course of the case will be good. The wound is rapidly closing, and the suppuration from the tympanum will be, and is being, diminished by the frequent irrigation employed. The only question remaining is how far the opening in the tympanum will close, and thereby the sense of hearing be restored. If the opening in the tympanum is closed by granulation tissue the hearing will return; if otherwise it will be impaired.*

This case is both typical and exceptional,—typical of its class as regards the local symptoms excited by acute suppuration in the mastoid cells, and the proper treatment required; but exceptional as regards the conditions antecedent to the affection of the mastoid cells. It is only very rarely that mastoid abscess occurs except as a result of chronic or subacute suppuration of the middle ear. Much the most frequently it is a complication of suppuration of the middle ear of long standing, often of many years.

In this respect mastoid abscess differs from

* Nov. 11th—The wound is quite healed, the discharge from the ear has almost ceased, hearing is returning, and the patient feels quite well.

another affection of the mastoid, also a frequent complication of disease of the tympanum, viz., inflammation of the periosteum. Mastoid periostitis, like mastoid abscess, may be a consequence of chronic inflammation of the middle ear; but it is much more frequently the case that mastoid periostitis arises in the course of an acute simple catarrh of the middle ear. Some degree of mastoid periostitis is nearly always associated with acute aural catarrh.

But though it is true that mastoid abscess is commonly the result of chronic suppuration of the middle ear, it is important to bear in mind that it may occasionally follow a *recent acute otitis media* which has commenced either as a suppurative or as a catarrhal process. There is, I think, some danger of this fact being overshadowed, if not entirely overlooked, by the frequency and fulness with which attention is nowadays, and quite rightly, directed to the seriousness of chronic suppuration of the middle ear, especially as regards its most grave results, namely, mastoid abscess, thrombosis of the lateral sinus, cerebral, cerebellar, and subdural abscess, and meningitis. I say *nowadays*, because you must know that it is scarcely more than twenty years ago that chronic suppuration of the middle ear, so commonly spoken of amongst the public as "a running from the ear," was very lightly regarded by the profession. People used to die from pyæmia, meningitis, cerebral and cerebellar abscess—all the direct result of this so-called otorrhœa—without a suspicion being aroused that the disease of the ear had been the real cause of death.

Now this term *otorrhœa* is a bad one. It does not define the source or character of the discharge; and it would be well to expunge it altogether from the nomenclature of ear diseases. It no more signifies the flow of pus than it does that of serum or blood; nor does it more signify a "running" from the tympanum, than from the external auditory canal. It has been the cause of much confusion of thought and frequent neglect of treatment, for it has been the means of delaying, and, in some instances, altogether preventing the distinction being drawn between suppuration of the external ear and suppuration of the middle ear, attended with perforation of the membrana tympani. Chronic suppurations of the auditory canal, even including the cases due to eczema auris, are comparatively rare; and yet patients with a chronic purulent discharge from the ear are often

supposed to be suffering from inflammation of the external ear, when the real seat of the disease is in the middle ear. It is this error that has led, and still does lead to a neglect of local treatment in chronic suppuration from the middle ear. A discharge due to eczema auris, to seborrhœa, or otitis externa will often get well under improved hygienic and dietetic conditions, and by the aid of cod liver oil and tonics; but these remedies do not suffice to cure a suppurative otitis media. It would be much nearer the truth, and much safer in practice, to take it for granted and treat it accordingly, that a chronic discharge of pus from the ear has its origin in the tympanum instead of on the outer side of the membrana tympani. There are probably more than a score of cases of suppuration in the tympanum for every case of chronic suppuration in the external auditory passage. St. John Roosa years ago pointed out that in the practice of several hospitals in the United States, chronic *suppuration* of the middle ear was three times more frequent than inflammation of the external auditory canal, including non-suppurative as well as suppurative diseases.

The usual form of acute suppuration in the external auditory canal is follicular in origin. The skin which is very tightly bound down to the perichondrium and periosteum of the canal contains numerous hair follicles, sebaceous glands and their follicles, and tubular ceruminous glands. When any of these follicles become inflamed they cause the most intense suffering; and when they burst, or are opened, they discharge a small quantity of pus. This is the commonest form of suppuration of the external ear.

But that is extremely different from the kind of discharge which takes place in middle ear disease, and which is always freer, and generally of chronic or subacute duration.

In eczema auris the source of the discharge is obvious, and is painless for the most part; the discharge ceases with the cure of the eczema.

There was, and still exists, an idea that it is harmful to stop a purulent discharge from the ear. This popular fallacy is due to the mistaken teachings of some of the older writers, especially of some of the older French writers. Sir William Wilde tells us "because it was observed that on the supervention of cerebral disease, discharges from the auditory canal have lessened, practitioners, mistaking the effect for the cause, have been led to believe that the sudden 'drying up' of the discharge

produced a metastasis to the brain, a notion as crude as it is unsupported."

There are still many cases on record, and the like are still to be met with in surgical practice in which an external flow of pus ceases, but not before the ulcerative process has spread beyond the limits of the tympanum, and has set up inflammation of the brain or its membranes, or its venous canals. Such cases, however, afford no more argument against treatment for the arrest of suppuration in the tympanum than the set of pathological changes known as "surgical kidney" which sometimes are hastened in their progress by the introduction of an instrument, afford an argument against the timely and judicious treatment of stricture of the urethra or enlargement of the prostate: or against the cure of gonorrhœa or gleet because we often find an epididymitis occurring simultaneously with the spontaneous cessation of the urethral discharge.

Another danger, it seems to me, is not unlikely to arise at the present time; and that is that we may lose sight of the importance of studying the beginnings of middle ear disease by the great attention and prominence which is given—and rightly given—to the surgical operations which are now employed in the treatment of the very severe, and, if left alone, fatal results of otitis media. It is a matter of the first importance that, owing to the genius and pioneering skill of a few modern surgeons the most brilliant results can be, and are, obtained by surgical operations upon the brain for abscess, and upon the internal jugular vein and lateral sinus for thrombosis,—conditions which formerly invariably ended in death, either from compression or pyæmia. But to the patients themselves it is a matter of still greater importance that the early beginnings from which such formidable and terrible conditions result should be recognized and cut short. Prevention is better than cure; and an early cure by simple agents is better for the patient than a great and dangerous operation after a lingering illness. There is not much *éclat* attaching to the application of a few leeches or to the use of the warm douche; yet these remedies alone are often sufficient to cut short an inflammation, and prevent the occurrence of suppuration in the tympanum. There is, on the other hand, much *kudos* attached to the successful issue of an operation performed for one of the very serious after-results of suppuration of the middle ear. The patient does not appreciate, because he cannot

realize, the degree of benefit conferred on him by the simple remedies I have mentioned in the one case; whilst the surgeon has the satisfaction of feeling that he has performed a brilliant and successful operation in the other. But the saving of pain as well as of life is the surgeon's mission; and his aim must therefore be to protect his patient from suffering and danger, rather than to permit the suffering to continue, and then to save the patient when he is on the very threshold of being lost.

Let it be admitted at once that there are cases in which the first and only intimation of any morbid action given by the ear is a discharge of pus from the auditory canal. Such was the case in the patient whose history I have related—with this exception, that he had had what he described as neuralgia at the back of his head. He had no symptom referable whatever to his ear, and therefore there was no indication for treating it. He had been working in a very draughty shop, but had felt no pain or fulness in his ear, nor had he suffered deafness or soreness of throat. He had always enjoyed good health, and never had a day's illness in his life. But one night he suddenly felt a noise in his right ear like an explosion, and from that time there has been a discharge from the ear. This was the first evidence of any ear trouble which he perceived. Had we had the opportunity of examining the ear at this time there is no doubt we should have discovered a perforation of the membrana tympani. As a matter of fact, the man was told at the Throat Hospital on the day but one after the discharge had commenced, that he had an abscess in the drum of the ear which had broken.

Thus we see that "ear-ache" does not necessarily nor always precede a "running from the ear." It is not probable that the abscess in this man's ear was confined to the layers of the drum-head till it broke into the external auditory canal; but even if this was the condition of things, the cavity of the tympanum must soon have been encroached upon, and have been the source of the very free discharge which continued for a month and three days.

It is generally thought that some symptoms, such as those of pharyngitis, or a sense of fulness in the ear, or a slight degree of deafness, precede a sudden and painless perforation of the membrana tympani; but in this instance, I repeat, the patient entirely denies their presence.

The suddenness of this case entitles it to be classed as *acute*. The suppuration was pretty certainly the consequence of an acute catarrh, due to the exposure to cold draught which he had for a week or two experienced in his workshop.

Let us now consider THE CAUSES OF ACUTE OTITIS MEDIA. The usual starting point of acute suppuration of the tympanum, is *inflammation of the naso-pharyngeal mucous membrane* induced by cold, or small-pox, scarlet fever, or measles; or one of those other affections which are so often associated with affections of the pharyngeal mucous membrane—diphtheria, tonsillitis, coryza, bronchitis, pneumonia, whooping-cough and typhoid fever, all play an important role in the production of acute aural disease. At first the disease is usually of the catarrhal variety, but too often it soon passes into the suppurative form.

The use of the nasal douche for the treatment of naso-pharyngeal catarrh, has also been known to excite acute otitis media. It is, perhaps, through the faucial extremity of the Eustachian tube, that acute catarrhal and suppurative inflammation is most frequently started; but the cause occasionally acts along the auditory canal through the membrana tympani, and not along the Eustachian tube,—as, for instance, when a draught of cold air blows on the side of the head, as in the case I have given you, or when cold water gets into the auditory canal, and, from being allowed to remain for some time against the membrana tympani, starts the otitis media.

Another cause of acute otitis media is *frequent bathing*, especially the ducking of the head in water and allowing the water to get into the auditory canal, and to rest against the tympanum without thoroughly drying the ears. I could relate to you the interesting case of a relative of one of the members of our staff. He had been bathing very frequently in the sea. One night he was suddenly seized with acute pain in the ear, which, continuing for three or four days, he came to London, and I was asked to see him. At that time he had, associated with the most acute pain in the ear, a considerable amount of redness and oedema over the mastoid bone, where pressure readily produced pitting, and gave rise to a sharp sense of pain. It was deemed right that he should be immediately treated by an incision over the mastoid process; and in the course of the next day this was done, and a small quantity of pus was let out from beneath the periosteum. From that

moment the patient lost all his pain, and recovered perfectly his hearing. In this instance, we had a case of catarrhal tympanitis started by frequent bathing; and associated with the tympanitis we had, what has in many other cases been observed, a considerable degree of periostitis of the mastoid. We had, indeed, what I was referring to in the earlier part of the lecture,—mastoid periostitis associated with acute catarrhal otitis media; and by the early incision down upon the mastoid and through the periosteum relief was given to tension, and the small quantity of pus which had formed being allowed to escape the reparative process was started.

Several other cases of the sort I have seen, where an incision down on to the mastoid bone, dividing the periosteum, has given relief to tension, and the patients have got well from the moment of that incision. Without there being any matter to let out, the mere removal of tension by the division of the periosteum is often quite sufficient to stop the progress of the mastoid periostitis. Though the common course is that the otitis media starts from the faucial extremity of the Eustachian tube from some affection of the naso-pharyngeal mucous membrane, yet you must remember that in other cases the course of the trouble is along the auditory canal.

Next let us consider the *symptoms of these acute affections of the middle ear*. Notwithstanding exceptions such as the above, the symptoms of acute catarrhal and acute suppurative media are usually most severe. A child will awake from sleep screaming because of the pain in its ear; adults are seized quite suddenly with the most intense agony, causing them to tremble and cry out. Probably there is no pain so intense as that which is excited by an inflammation in this little space—the tympanum. Patients have been known to lose their reason on account of it; children have been supposed to be affected with acute disease of the brain or its membranes. This pain comes on suddenly, and at once attains to its highest pitch of intensity, and very rapidly suppuration is established. In some cases the sensations of fulness and of noises in the ear are most distressing, and great tenderness is evinced by pressure over the tragus. The membrana tympani is seen to be in a state of vascular injection, and varies in colour from a pinkish hue to an intense red. Sometimes, instead of an impairment of hearing there is increased acuteness, and frequently

with the ear symptoms there is associated from the beginning the tenderness over the mastoid due to the presence of mastoid periostitis. Febrile symptoms are almost always present. The temperature is often raised three or four degrees, or more; the skin is correspondingly dry, and the thirst intense. Delirium is sometimes present, especially in young persons; the head throbs, and the various secretions are seriously disturbed, as you find them to be in all febrile action.

The only symptom complained of by my patient before the discharge began was neuralgia in the back of the head, which ceased entirely after the running was established. Neuralgic pains have often been noticed in other cases, and in quite the first stages of middle ear disease; and they have led to the mistaken diagnosis of neuralgia, and to the useless treatment—for it is useless in these cases—by Quinine and Opium, which is the proper treatment for many cases of neuralgia.

A few words as to the *treatment* of acute otitis media. Instead of ignoring the importance of "ear-ache," or standing helplessly by and allowing this "ear-ache," which is no other than acute catarrh of the middle ear, to run on to suppuration, and thence to mastoid abscess, thrombosis of the lateral sinus, or brain abscess, we should at once employ certain means, which, when early and properly used, often act like a charm, and produce effects in their way as brilliant and satisfactory as the most successful operation in the later phases of the disease. These means are *local blood-letting* and the use of *the warm aural douche*. It has been often noticed that when a child is screaming with pain in the ear the application of a couple of leeches upon the tragus has been the means of relieving the pain, and the child has gone off to sleep. This early application of leeches for the treatment of otitis media of the catarrhal form is very efficacious, and should certainly be employed.

When leeches are not at hand, or you think it for any reason undesirable to employ them, the next most efficacious thing is the continued application of the warm douche. It is not desirable to use a syringe and inject warm water into the part; but with an extemporised irrigator, such as an indiarubber tube dropped into a jug of water, you can get a syphon-like action and a free flow into the ear, which, in the course of a few minutes, will produce a marvellous alleviation of the condition. This, frequently repeated, is often quite

sufficient to stop the disease and start the process of repair.

It is of very little use to give Opium internally to allay the pain; it does not produce much effect, but the warm douche is of considerable use in relieving pain.

Other things should be attended to, such as the treatment of the naso-pharyngeal mucous membrane. A gargle of an antiseptic and astringent character should be used, if there is any affection of the naso-pharynx. Borax, Chlorate of Potash, Alum, or some drug of that kind, in a gargle is often of the greatest value in removing the cause which has started the middle ear disease. Externally, fomentations should be applied to the neck.

In some cases paracentesis of the drum membrane, when that is found to be bulging, is very efficacious and may at once relieve pain.

It will be noticed that the discharge which at first ran from this man's ear was of a red colour, but afterwards became paler, and then quite purulent. Without having seen this fluid it is impossible to say whether it was pure blood, or to what extent it was admixed with blood. Probably it was pus mixed with blood which escaped from a vessel of the membrana tympani, which was ruptured at the time the drum membrane gave way. But you ought to know that there is a condition described as *otitis media hæmorrhagica*. St. John Roosa, for instance, has recorded two cases of very acute aural catarrh in which the membrana tympani was perforated without suppuration, but with an abundant discharge of blood. He states further, that in many cases of paracentesis of the tympanic membrane, for the relief of inflammation of the lining membrane of the tympanic cavity, blood alone escaped. It would seem from the course of the cases which he and others have recorded that this hæmorrhagic form of otitis media is much more tractable than the suppurative inflammation of the middle ear.

You should know, too, that there are also well recorded instances of hæmorrhage from the tympanic vessels into the middle ear, occurring in the course of Bright's disease, just as hæmorrhage takes place from the retinal vessels, which have become atheromatous from kidney affection; so that in a case of Bright's disease, when there has been no exposure to cold, and nothing wrong with the naso-pharynx, if there should occur a sudden attack of pain in the ear, you may bear in

mind the possibility that it is due to distension of the tympanum by blood from diseased vessels which have ruptured.

The mastoid abscess which formed in the man whose case we are considering, occurred a month and three days after the first onset of the discharge from the ear. This is undoubtedly a somewhat short time. In most cases the abscess occurs after many months, in some cases after many years. But the acuteness with which the mastoid affection set in, and the amount of pus we found there only four days after the commencement of the pain in the mastoid region, show that the process was a very acute one in the mastoid cells, and that had treatment not been at once employed, we should very rapidly have had destruction of the whole interior of the mastoid bone, and probably the disease spreading on through the mastoid cells to the lateral sinus. This is the way in which the lateral sinus becomes thrombosed, namely, by the extension backwards of disease to the hindermost parts of the mastoid cells, which lie in close proximity to the lateral sinus.

Now it may possibly occur to you to ask, How is it that the pus does not empty from the mastoid antrum into the tympanum, and thus out through the external auditory canal? It might also occur to you to ask, How is it that in acute suppurative otitis media, when we have the tympanum filled with pus, it does not escape down the Eustachian tube? Well, a mechanical explanation serves in both cases. The Eustachian tube does not open at the lowermost part of the wall of the tympanum. Thus there may be a considerable accumulation of pus in the tympanum, which cannot possibly make its exit through the Eustachian tube; and again, the cells of the mastoid are, many of them, below the level of the usual communications between the mastoid antrum and the middle and external auditory passages. It thus becomes obvious that in order to give exit to this pent-up matter in the tympanum and cells of the mastoid, we must irrigate the tympanic cavity, and apply the trephine to the mastoid cells.

Now, Gentlemen, with regard to the position in which we should apply the trephine. I have had this bone [a divided temporal bone was shown] brought in for you to see the cells of the mastoid and their relation to the tympanum and external auditory canal. Notice the level of the external auditory canal, and you will see that many of the cells of the mastoid are quite a third of an inch

below the middle and external ear, so that they cannot possibly drain into the cavity of the tympanum or the auditory canal. This bone also serves to show us the point at which we should apply the pin of the trephine in order to give exit to matter in the mastoid antrum, and that best spot is at the level of the external auditory meatus and a little behind it, the trephine to be directed a little forwards as well as inwards. Thus we shall open directly into the middle of these various cells of the mastoid, and thus have a good and direct opening for the exit of the matter which is contained therein.

With regard to this operation there are certain accidents which might arise.

If you apply the trephine too far back beyond the ridge that runs up from the apex of the mastoid you would go directly into the lateral sinus. You are always easily able to feel in every mastoid its posterior ridge, a process which runs upwards from the apex of the mastoid. If you keep in front of that ridge and direct your trephine forwards and inwards you are safe as against the lateral sinus; but if you go behind that ridge, over it, or even in front of it, and direct your trephine backwards or straight inwards you run great risk of wounding the lateral sinus, and if the lateral sinus does not happen to be thrombosed you will get a very violent hæmorrhage as the result of its injury.

I want, also, just to mention to you that you may get sometimes very alarming hæmorrhage, even when the trephine is applied in the proper place and directed forwards and inwards. I could refer you to a case which happened to me in 1885, where a boy æt. 8, was brought under my care for inflammation of the mastoid process. It was inflammation really of the mastoid periosteum. There was no abscess in the antrum, but we were not quite sure of that, and therefore the mastoid periosteum was divided and peeled back a little way, and the surface of the mastoid bone inspected. No matter came from beneath the periosteum, so I thought it right before sending him back to bed to drill into the mastoid cells. I did this, using not an instrument so large as a trephine, but a small drill applied on a level with the external meatus, and a little behind it. I directed the instrument forwards and inwards; and when I withdrew the drill the most violent hæmorrhage occurred. The bleeding was furious. Pressure with sponge after sponge did

not check it. It was quite certain that from the direction and position of the drill, I could not have reached the lateral sinus; and I was persuaded—and other cases of which I have read, notably one by Sir William Dalby in the “Medico-Chirurgical Transactions,” support me in the supposition—that I must have wounded some small vein of the mastoid in direct and short communication with the lateral sinus. We know that if we wound a branch artery near a main trunk, we get bleeding from that small artery almost as furious as if we had a wound of the trunk itself. I think it is probable, indeed it seems to me to allow of no other explanation, that I wounded some small vein in the mastoid bone, which from its proximity to the lateral sinus, bled almost as furiously as a wound of the lateral sinus would have done.

Where the lateral sinus has been opened up by the trephine you have no alternative but to plug the opening, and trust to the formation of a coagulum to stop the hæmorrhage. But in this instance, as I had only a small opening made by a drill, I thought I would resort to some more secure and permanent measure than plugging with any antiseptic dressing or sponge. It occurred to me, therefore, to use a cribbage peg. This I obtained from one of the residents' rooms. Temporarily plugging the opening with a probe, I filed down the cribbage peg until it was of sufficiently small size to pass into the drill-hole. With sharp pliers I then nipped it off level with the mastoid and left that little bit of bone in the mastoid process of the child. We had no further trouble from the hæmorrhage; the whole of the child's pain in the ear and about the mastoid ceased from that time, and the boy got perfectly well. Although the operation was done in 1885 I occasionally see that boy now, and he is none the worse for his cribbage peg in the mastoid. It was a useful way of employing the peg, and served the purpose most efficaciously. It is also satisfactory to know that whilst he was relieved of pain by the division of the periosteum, he did not suffer any pain from the filling up of the hole I made into his mastoid process with this hard foreign body. I think that his trouble was due to the periostitis, and that I need not really have gone further than the division of that membrane. Other cases have convinced me that the division of the periosteum is often quite enough, and that unless you see, as we did in this case, some little tiny carious spot in the mastoid bone, it is not necessary to push the opera-

tion further than the free division of the periosteum.

I have used this case as the text for these remarks, Gentlemen, not so much to bring under your observation the operations which are employed to combat these serious results of otitis media as to draw your attention to the earliest symptoms of otitis media. I am quite sure that if you will pay attention to the early symptoms of acute catarrhal otitis, if you will address your treatment to them, and if you will also always bear in mind that a “running from the ear” is a matter of considerable importance, and that any ear from which there is a discharge going on ought to be carefully and frequently douched, you will prevent your patient getting any of those serious consequences which operative surgery has in these latter days done so much to relieve and cure.

Pay great attention to the early symptoms of what are called “ear-ache,” and also to “running from the ear,” and thus prevent the serious consequences which will ensue from the neglect of either of them.

A LECTURE

ON

PELVIC HÆMATOMA AND HÆMATOCELE.

Delivered at Charing Cross Hospital, October 26th, 1893,

BY

AMAND ROUTH, M.D., B.S., M.R.C.P.,

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(Concluded from p. 64.)

SYMPTOMS OF PELVIC HÆMATOCELE.

The best way to tackle this part of the subject will be to describe a case of tubal gestation which is the commonest cause of intra-peritoneal hæmatocele. We will assume that a patient has had some chronic salpingitis, as a result of which the tube becomes denuded, at the uterine end, of cilia. The ovum, escaping from the ovary at or about menstruation, becomes impregnated. Of

course the spermatozoa can pass up without cilia ; but the ovum, unendowed with spontaneous mobility, has to be carried down by the cilia (or by the current induced by them), so that on reaching the point where there are none it stops there and becomes implanted. The uterus sympathetically enlarges as if the ovum had got into its own proper cavity ; and for a month or so everything goes well. The patient probably thinks she is pregnant, or thinks nothing about it. She has no pain for the first few weeks. Then she generally has a little pain (tension) on one side ; and at or about the tenth or twelfth week rupture ensues. Uterine hæmorrhage may occur about this time, and the passage of a uterine decidua is pretty constantly observed.

Rupture may have occurred downwards into the broad ligament, or, as we are assuming now, into the peritoneal cavity. The probability is that it takes place over the placental site, so that part of the placenta bulges through the opening ; and generally the opening is sufficiently large to allow the foetus to escape. If both the ovum and the patient live, the placenta remains attached where it was, and any part of it which is detached becomes attached to any surrounding viscus—to the back of the broad ligament, to the uterus, bowel, or omentum ; and, occasionally, we come across cases where it is found to be solely attached to the surrounding organs, having transplanted itself, not bodily but gradually, from one part to another. The foetus may go on growing, and at full term an attempt at labour occurs ; and then, generally, the foetus dies, and may either suppurate or remain as an encysted lithopædion.

If, at the time of the rupture, the hæmorrhage is severe, it produces immediate shock—shock partly due to the rupture, partly to the hæmorrhage. Marked symptoms of internal hæmorrhage occur—syncope with pallor, sighing, sometimes sickness, the arms thrown about, great restlessness, and so on. All these would indicate a good deal of intra-abdominal hæmorrhage.

If one were to examine the patient then, one would find the abdomen slightly swollen, exaggerated possibly by retention of urine, which we at once relieve by the catheter, so that the amount of swelling would be lessened. Per vaginam very likely we should also make out very little. Blood recently extravasated is not palpable to the examining finger at all, if it is diffused throughout the abdominal cavity. Perhaps a slight sense of resis-

tance is felt, but there is no bulging down of Douglas's pouch, as there is when it is roofed over by adhesion. The abdomen will allow a woman to bleed to death into its cavity owing to its distensile powers.

If, however, the hæmorrhage is encysted by adherent bowel or pre-existing retro-uterine perimetritis, for instance, then it would form a mass in Douglas's pouch, at first elastic and more or less tense, but after a few hours getting a great deal harder.

The signs, therefore, vary entirely according to whether the blood is encysted or non-encysted, and the gravity of the prognosis can be similarly estimated.

After a few days, supposing the patient survives, the mass in Douglas's pouch becomes very hard ; and after a few days longer, instead of being perfectly rounded, it becomes more conical, tending to correspond in size and shape to Douglas's pouch, and gets more lobulated, owing to absorption going on in some places quicker than at others. Pressure on it is generally quite painless to the patient, differing in this respect from a retro-uterine perimetric phlegmon. The uterus, in cases of hæmatocele, is almost always pushed upwards and forwards, so that sometimes one cannot feel the cervix at all. It is fixed. By the abdomen one can feel generally the uterus above the pubes, and very often a large mass up to the umbilicus. This is the case when the hæmorrhage has displaced the intestines, and come bulging up along the back of the uterus much in the same way that a fireman's helmet covers the fireman's head, embracing it somewhat and coming over to the front, forming there a large lump sometimes reaching above the umbilicus, evidently continuous with the mass in Douglas's pouch. The hæmatocele is very rarely in the utero-vesical pouch, though one or two cases are described (Schröder) as having occurred in cases of hæmatometra.

The risk, as I have said, is entirely a question of whether the hæmorrhage goes on or not. If the lumen of the vessel that has been ruptured is a large one, or if another bit of placenta becomes detached, there is more hæmorrhage, and the danger to the patient is increased ; but the risk of hæmorrhage depends more than anything else on the question of whether there is sufficient resistance, that is to say, whether the hæmorrhage is encysted or not.

If the patient recovers, absorption generally

takes place. Suppuration may, however, ensue from septic organs being in the neighbourhood; or, as I have said, if it has been a tubal gestation, the foetus may go on growing to the full term.

The diagnosis has to be made from, I think, only two things. The first is an acute retro-uterine perimetritis. Here there is no symptom of a sudden onset other than a rigor. There is generally pain for a day or two before this. The commonest cause of retro-uterine perimetritis is a spreading of inflammation up from the vagina or uterus, so that there is generally pre-existing trouble of some sort; or there is a history of sudden check of menstruation from a chill, with, generally, an initial rigor, and the pain is extremely well marked. The knees are drawn up, the abdomen becomes tympanitic and extremely painful, and the temperature is high, whereas, in pelvic hæmatocele it is at first subnormal.

The physical signs are different. With hæmorrhage, supposing it is encysted, there is a large mass behind, and there is no, or very slight, tenderness. In cases of perimetric phlegmon the tenderness is very severe, and there is more or less induration to be felt even at the onset, at first, somewhat out of easy reach near the fimbriated end of the tube, but soon to be felt at the bottom of Douglas's pouch or in one or other posterior quarter of the pelvis behind the broad ligament, but never on the first day forming a large mass as it would if it were hæmatocele.

The other thing one has to diagnose hæmatocele from is impacted retroversion of the gravid uterus. Here there is no pyrexia, but if the retroversion is suddenly induced in the course of pregnancy, the suddenness of the onset is something like that which would occur in the case of rupture of a tubal gestation. The shock and anæmia would be well marked in hæmatocele, and the vulvar mucous membrane would be pale instead of the bluish tint of retroversion of the gravid uterus. Except where there is also retention of urine, there is no abdominal tumour in retroversion of the gravid uterus; whereas, in severe hæmatocele there is an abdominal tumour in almost every case. Then the finger being passed per vaginam detects a large mass in a retroversion of the gravid uterus bulging downwards between the rectum and vagina at a much lower level than a hæmatocele, and not nearly so hard. It is boggy and capable of some mobility. With one finger in the rectum, and a finger of the other hand in the vagina, one can distinctly make

out that the tumour is compressible to some extent. Sometimes one can make out it contains fluid, and sometimes an intermittent contraction can be felt. If the cervix can be felt at all, it can be traced down backwards into the retroverted uterus; whereas, in hæmatocele, bimanual palpation, or the use of the sound would show that the cervix was continuous with the abdominal tumour there.

It is important, of course, to make the diagnosis, because otherwise one would be trying to reduce a hæmatocele, as has actually occurred, believing it to be a retroversion of the uterus.

TREATMENT.

The dictum used to be that in every case of intra-peritoneal hæmorrhage you must open the abdomen; and that was for some time the accepted doctrine. But unquestionably a great many of these cases do recover; and it depends, as I have said, entirely on whether the blood is encysted or not. If the patient is evidently losing ground, or if there has been a recurrence of hæmorrhage and you are unable to make out a definite tumour in Douglas's pouch definitely outlined in the abdomen, you may be pretty sure the blood is non-encysted; and in that case it becomes one's duty to save the patient's life. Choose your own time, and let her rally if the hæmorrhage does not seem to be going on. When the abdomen is opened, blood wells, out, and hæmorrhage is apt to recur when pressure is thus lessened. Therefore, the incision should be a fairly large one, three or four inches. No time should be lost in sponging out blood-clot, but go at once to the fundus uteri, draw your fingers along the two tubes; feel for the lump on one or the other side, and, with Spencer Well's large forceps by preference, you clamp the ligament on each side of the lump, and then the hæmorrhage is entirely under control, the ovarian arteries and veins being thus secured. You can then remove the blood-clot, and rally the patient by a saline infusion, for instance, and take your time about tying the tube and broad ligament on each side of the rupture and remove the gestation sac bodily, being careful to look about for a foetus, which is generally found amongst the clot. Sometimes the patient is so low that you dare not operate without rallying her; and the best way is by the saline infusion. Of this you may inject large quantities into the rectum, from which it is rapidly absorbed in these cases; or better still, if

you have an apparatus, into a vein in the arm. Use a teaspoonful of common salt to the pint of boiled water which has been allowed to cool down to the temperature of the body. If it is to be injected into a vein, all that is necessary is a funnel with an indiarubber tube and a glass nozzle, or any nozzle that will go into the vein. The funnel is raised or lowered according to the pressure you want, and sufficient water is passed in. Three or four or even more pints can then be passed into a vein. If, however, the patient seems to be steadily rallying, and especially if the hæmatocele becomes encysted and stationary after a day or two, it is much better to leave her alone, avoiding any interference.

A CLINICAL LECTURE ON THE SURGICAL TREATMENT OF LUPUS VULGARIS.

By WILLIAM ANDERSON, F.R.C.S.,

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the Department for Diseases of the Skin.

THE many examples of lupus that have been demonstrated from time to time in the wards and out-patient rooms, have familiarized you with the characters and progress of the disease. They have shown you, too, that although it is cruel and intractable enough to deserve its ill-omened name, there is nothing less wolfish than its unobtrusive mode of attack and the insidious patience of its insatiable ravages. The *Canis lupus* is undergoing extinction before the march of civilization, but the *Morbis lupus* thrives most in the very centres of our progress, defying all the resources of preventive science, and opposing a grim and tenacious vitality to our efforts to destroy it.

We have had little reason to be proud of the results of our efforts to cure the disease. Not a single known drug in or out of our pharmacopœia exerts any specific influence when administered internally; and not one of our chemical agents gives a certain promise of extirpation. Recently a new remedy, based upon the most advanced researches in bacteriology, the "tuberculin" of Koch, raised strong hopes, but ex-

perience has so completely failed to realize them, that I do not think it necessary to go into the details of the method or the history of our disappointment. It will be sufficient to say that the experiments of Koch have failed in their immediate object, but, like those of the old alchemists, they may still prove to contain the germ of important results in the future. He has given us a preparation having the remarkable property of setting up a powerful reaction in the very focus of the disease, and, although it has hitherto proved too little under control to offer safe or permanently good effects, the very fact that such a localized reaction can be voluntarily obtained, is pregnant with suggestions and possibilities. In the meantime the method lies outside the region of practical therapeutics.

Another form of treatment may be considered in association with that of Koch: the inoculation of the seat of disease with erysipelas. It has been noticed from time to time that when a patient suffering from lupus contracts erysipelas the acute infection has a great influence upon the chronic disease, often arresting its progress, and sometimes causing its complete disappearance; and it has been proposed to introduce the erysipelatosus poison into the system artificially. This suggestion, however, like the last, has been weighed and found wanting; for the remedy, especially when applied to the most usual seat of lupus, is by no means free from danger, and the good effects, when secured, are rarely sufficient in extent or duration to justify the risk. The tubercle bacilli, assuming that they are the cause of lupus, may be scotched, but are not killed, and the new germs may prove even more dangerous than the enemy they were commissioned to destroy.

We are driven, then, to treatment of a purely surgical character, but of course we must not neglect to support our local measures by all the constitutional resources that each individual case appears to demand. Fortunately, the local treatment has been rewarded, in later years, by a considerable measure of success. It aims at one of four objects:—To destroy the disease by the direct action of chemical or thermal agents; to change the character of the morbid structure by minute subdivision (scarification) with a cutting instrument; to scoop out (erosion) the lupous elements as completely as possible from their bed by means of a suitable instrument; or

to excise the new growth, with as much of the surrounding tissues as may be deemed advisable.

The oldest destructive remedies are *caustics*; and of the multitude of forms that have been tried the most useful are Arsenic, one of the oldest, and Salicylic Acid, one of the most recent; because these, when suitably applied, tend to destroy the unstable lupous growth, while the healthy tissues resist their action. The Arsenical Paste recommended by Hebra is a trustworthy formula, and Salicylic Acid may be conveniently applied in association with Creasote, which mitigates the pain of the caustic and is, perhaps, useful in other ways. Pyrogallic Acid has also been strongly advocated, but I have little experience of its value. On the whole, however, these applications give scant encouragement. In a small proportion of cases, chiefly those of a mild kind, they are temporarily successful, and occasionally effect a permanent cure, but more often they fail to do good, and the irritation for which they are responsible has at times appeared to hasten the spread of the disease.

Of the indiscriminating caustics, such as Nitrate of Silver, Chloride of Zinc, Caustic Potash, and the like, there is little good to be said. They destroy healthy and diseased tissues alike, and are liable either to act too deeply, producing even more loss of substance than the knife, or they fail to kill the whole of the lupus, and may stimulate to a more rapid growth the morbid elements that are left. They may, however, be useful as adjuncts to other treatment, but as a means *per se* their past does not give much promise of an honourable future. The same may be said of the cauterizing, actual or thermal, which still has its supporters.

The treatment by *scarification* is applicable to limited areas of disease in regions where the character of the scar is of especial importance. The operation is effected by means of a sharp knife. An ordinary scalpel serves the purpose well, or a special instrument, consisting of a number of parallel closely-set blades, may be used instead; the single knife has, however, the advantage of conveying a more exact impression of the resistance encountered, and is altogether more efficiently manipulated, if somewhat slower in action. The knife held with the edge at right angles to the surface is made to "cross-hatch" the diseased tissue as in shading a pen-and-ink drawing, subdividing the cell-growth into a number of minute

segments, each of which is thus isolated from the rest and from the main vascular supply, down to the healthy structures beneath. It is obvious that the tactile sensibility and manipulative experience of the surgeon must to a large extent govern the result. Each incision should go down as far as, and slightly into, the firm bed of fibrous tissue, and the "mincing" must be as fine as possible. After the process is completed I have found the immediate application of a 2½ per cent. solution of Carbolic Acid to the area acted upon, lends a valuable addition to the effect. But the process is painful and sanguinary, and for many patients it is both exhausting and alarming. Local anæsthetics are seldom of use, and unless a general anæsthetic be administered only a very small area can be dealt with in a single sitting, and the process of treatment becomes very tedious as well as unpleasant. In the most favourable cases, the result is often very satisfactory, as the scar is smoother and more uniform than after the use of caustics, and does not appear to have the tendency to cheloid hypertrophy that is apt to follow erosion. Given a suitable case and a courageous subject, the plan is worthy of a trial. One of the patients of whom I am most proud, submitted to a treatment extending over several months with heroic determination, and the disease, which had involved the whole of one cheek and the corresponding side of the nose, was replaced by a scar that is scarcely to be distinguished from the adjacent healthy skin. Too often, however, the desired end is never reached, and the disease, after delusive promises of cure, resumes its hold upon the tissues; but as it does not prejudice further treatment it may be tried for patches of limited size confined to the nose and the adjacent part of the cheek where caustics and erosion leave scars of a more conspicuous character.

The removal of the disease by the curette or knife gives a more certain prospect of cure than any of the measures I have yet described. Of the relative merits of the two methods of removal a great deal might be said, but it will be found that each has its advantages in special cases.

Erosion is more thorough and speedy than scarification, and more rapidly selective than the best caustic agents, but it is far less radical than excision. There are, however, certain cases in which it is preferable to the latter, such as those affecting a large area of the face and neck, where the use of the knife would involve a wound of serious extent

and no little danger. In such examples the curette manipulated with boldness and discrimination may nearly always be relied upon to get rid of the greater part of the disease, and occasionally, of the whole; and the surgeon watching for the marginal nodules that are almost sure to appear sooner or later, is in a position to use the knife with effect and without danger. Two of the cases now before you are of the kind alluded to, and have been successfully treated by erosion, followed by excision of the recurrent nodules. They have now been free from the disease, the one for a year, the other for nearly two years, and the result has only one defect, the presence of some cheloid hypertrophy of the scar, a fault which I propose to deal with by a final operation.

The instruments employed are Volckmann's sharp spoon, and the ring curette. The former is preferable for the treatment of small foci of lupus, the latter where large areas have to be traversed.

The value of the curette depends wholly upon the judgment and energy with which the instrument is wielded. It is useless or worse than useless to scrape away only the more superficial portions of the disease, and yet it is to be feared that this is all that is done when the method falls into timid or unpractised hands. The cutting edge should be carried well down to the firm fibrous bed which surrounds the new growth, and during the scooping process must be pressed forcibly against the resisting tissue, particular attention being paid to the raised and often undermined edges where the activity of the morbid process is greatest. The result is a smooth, sharply defined depression, larger than the appearance of the lupous patch before treatment would have seemed to promise, and bleeding somewhat freely from the small supplying vessels which have necessarily been torn in the course of erosion. The hæmorrhage ceases in a few minutes, and we may now either leave the surface to heal after dusting it with Iodoform or Aristol Powder, or we may attack the fibrous bed with a supplementary caustic.

If we decide to use a caustic, we may employ one of the selective agents, Salicylic Acid by preference, applied in the form of Unna's plaster, or in combination with Vaseline; but the regulated use of one of the stronger caustics is perhaps more certain in its results. Chloride of Zinc Paste, Nitrate of Silver, and Potassa Fusa, are those most in favour. Of these I have found the last most

satisfactory, because it is the most searching, the most manageable if carefully used, and, unlike the other two caustics, leaves no slough to undergo a tedious process of separation. I apply it by rubbing lightly over the central portions of the scraped area, more strongly at the margin from which extension of the disease was going on; and after a few moments—the time varying with the effects desired—the action is arrested by pressing pads saturated with Acetic Acid over the part until the alkali is neutralized. The surface is then dusted over with Iodoform, which combines with the blackish exudation left by the chemicals, and forms an aseptic scab under which the healing process goes on securely. No dressing is required in ordinary cases, but where the area is large, a layer of alembroth or other medicated wool may be applied and held in place by a bandage.

The effect is nearly always satisfactory, although it frequently falls short of completeness. For a while the whole of the newly cicatrized area appears sound, and in fortunate cases a permanent cure may have been obtained, but more frequently, while the central area remains sound, the active segment of the margin sooner or later shows signs of renewed disease, and if the warning be neglected, the surgeon's labour may be entirely wasted. Once the returning growth is declared, no time should be lost in removing it—not by erosion, but by excision.

Unfortunately, the scar left by erosion is by no means an ideal one—usually it is more or less irregular, and in places deformed by ridges of cheloid hypertrophy. These ridges, otherwise innocent, may be very unsightly; and although they lose the dull reddish tint which makes them more conspicuous in their earlier stages, they seldom disappear. The appearance may be greatly improved, however, by shaving off or excising the more obtrusive elevations with a sharp scalpel and allowing the cut surface to heal under Zinc or simple ointment; or, still better, to cover it with a small epidermic graft.

The sheet anchor of surgical treatment, *excision*, I have left until the last. For the last twelve years I have employed the knife in every case where it was not objected to by the patient or contra-indicated by the situation and extent of the lesion. The plan is especially suitable for small areas about the face and neck, and for areas of any ordinary size—not exceeding 5 or 6 inches in diameter—upon the neck and limbs, but it is possible

that it might be applied in even more extensive disease in certain cases. The results have been almost uniformly excellent; the scar can usually be made smaller and more presentable than that left by any other method of cure or by spontaneous healing, and the cure is nearly always permanent.

The excision is effected with a scalpel. In my experience it is rarely necessary to carry the section far beyond the margin of the growth. I seldom allow more than an eighth of an inch except opposite the margin at which extension has been taking place, and even there from a sixth to a quarter of an inch will be found ample. Moreover, I do not carry the knife deeper than the superficial fascia. It is possible that some infective elements are still left, but the active process of repair appears to be able to dispose of them in some way or other, for I have never seen a recurrence. There is indeed little doubt that in lupus, as in tubercular arthritis, operation seldom removes every germ of disease, but the results in both cases are none the less admirable and lasting.

The disease taken away, the area of cicatrization may be lessened and sometimes reduced to a line by sutures, sub-epidermic sutures being always employed upon the face and other parts where the scars of needle holes would be objectionable. When the wound cannot be entirely closed, the surface left may be covered with large epidermic grafts, after the manner of Thiersch, or in certain cases by a plastic operation.

In conclusion I would urge you to begin local treatment as early as possible; to use the most radical measures that the circumstances will permit and justify; and to fight incessantly against discouragement, remembering that there are few cases in which perseverance will not gain its reward.

THERAPEUTICAL NOTES.

Olive Oil for Lead Colic.—The *Gazette Médicale de Paris* contains Combemale's report of cases of lead-poisoning treated medically by large doses of Olive Oil. For attacks of colic, oil was given by the tumblerful with most gratifying results. Fifteen drachms of Olive Oil a day were ad-

ministered in chronic lead-poisoning, with a resulting diminution in the tremor, in the difficulty of standing and walking, and all the nervous symptoms. The observer states that it is impossible to say just how Olive Oil acts upon lead. The fact remains that doses of fifty drachms will cure lead colic, and daily doses of fifteen drachms will relieve many of the most distressing manifestations of plumbism.

Lozenges for Phlegm in Bronchitis, Royal Hospital for Diseases of Chest, City Road.

R	Pulv. Acaciæ	3ij
	Pulv. Sacch. Alb.	3j
	Pulv. Tragac.	3ij
	Extr. Glycyrrh.	3iij
	Olei Anisi	℥℥

M. Gt. Troch lxxx. One to be sucked occasionally, especially if the phlegm is tight.

REVIEW.

The Student's Handbook of Gynæcology. (E. and S. Livingstone, Edinburgh, 1893.)

This little book aims at brevity and conciseness, and makes no pretension to compete with the exhaustive gynæcological text-books. It commences with a short account of the anatomy of the female pelvis; and then gives brief accounts of symptoms, causes, and treatment of various common disorders of women. It contains 49 engravings, reproduced from various works. Both they and the printing are excellent, and the book will be found very useful as an introduction to the important subject with which it deals. It is in every way vastly superior to most books of this kind.

We regret that owing to pressure on our space this week we are compelled to hold over Dr. Boxall's Lecture II. on "The Use of Antiseptics in Midwifery" until next week.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 6, 1893.

A CLINICAL LECTURE ON THE VALUE OF PHYSICAL SIGNS.

Delivered in Guy's Hospital, Nov. 11th, 1893, by
FREDERICK TAYLOR, M.D., F.R.C.P.,
Physician to the Hospital.

TO-DAY, Gentlemen, I have undertaken to make some remarks on The Value of Physical Signs. In the wards we have to interest ourselves in physical signs, and the question of their value frequently comes before us.

By "physical signs" we may be held to mean "the phenomena ascertainable in the chest and abdomen by the use of the eye, the ear, and the hand." I do not feel sure that that is a complete definition of "physical signs," but it is what in a general way we mean by that expression.

Now the value of physical signs may be considered, (a) *in relation to other means of forming a diagnosis*, to other means such as the consideration of the pulse, the consideration of the temperature, the consideration of the history, and the consideration of the complaints of the patient; and you may ask, what is the relation of the physical signs in any particular case, to these other means?

It often occurs to me to remind myself of the observation that one of my late colleagues made on one occasion, namely, that having once determined that you had before you a case of acute pneumonia, you never need examine the case again. That, of course, may seem to be carrying the matter a little too far; but what it really means, no doubt, is this—that having once ascertained a case to be one of pneumonia, your estimate of the condition of the patient and his chances of recovery, and your guides for treatment, are derivable almost entirely, if not entirely, from the condition of the pulse and the condition of the patient's nervous system; that is to say, whether he is delirious, or whether he is re-acting in one or other way to the disease which you know he is suffering from, and that whether the pneumonia is extending a little in this way or a little in that, is of very slight importance as compared with these other indications. It is an expression also of what, I suppose, is now

fully recognized, that pneumonia is rather to be considered an infective disease, of which the local conditions are simply manifestations, and of which the local conditions are not the prime cause, and that it is not an inflammation of the lungs starting a general symptomatic or inflammatory fever. We know perfectly well, also, that in pneumonia the physical signs indicating consolidation of the lung very often persist for days after the fever has subsided, and when there cannot be any doubt but that the patient is practically out of danger so far as the acute stage of the pneumonia is concerned.

Now, some qualifications may fairly be asked for in connection with that formula, especially in relation to the possibility of the development of a pleurisy and an empyema upon a pneumonia. The development of an empyema secondary to a pneumonia occurs every now and then; and that is a very good reason why one should rather critically examine the lung from time to time. But the pneumonia once ascertained to be a pneumonia, may, so far as physical signs are concerned, sometimes be quite safely left alone; though, of course, I do not recommend it as a routine practice. On the contrary, we ought to know all we can of every case.

The value of physical signs, again, may be considered, (b) *in relation to each other*. The value of one physical sign may be considered in relation to the value of another. We may ask whether one is a more trustworthy or reliable means of getting at the condition of an organ or stage of a disease than another.

With regard to this last way of viewing things, it seems to me that to discuss it at all fully would be to go over the whole ground of the subject of percussion and auscultation as it is dealt with in various treatises by Gee, Skoda, Vierordt, Graham Brown, and others, and it would be almost impossible, I think, to deal with that in one lecture, except in the most superficial way.

But with regard to the importance of physical signs in relation to other means of investigation, I think there are some general principles that may usefully be touched upon.

I think the most important thing to bear in mind—and it is a fact that perhaps a good many of us do not always get hold of at once—is that

physical signs must be looked upon as indicating not any one particular disease, but only some particular condition of the organ which is being investigated; and if a particular physical condition can be brought about by different diseases, then the physical signs will not tell you what the disease is of themselves; but you will only arrive at that knowledge by a consideration of other means of observation, or other facts in the history or condition of the patient. It is of course true that we speak, for instance, of the physical signs of phthisis, the physical signs of pneumonia, and the physical signs of pleurisy; but we know perfectly well that a particular physical sign may be present both in phthisis, and pneumonia, and perhaps in pleurisy; and that as a means of diagnosis one cannot positively rely upon the physical sign itself.

I have not said—perhaps you do not want the question raised—exactly what one means by the *physical condition* of the lung as contrasted with the *disease*. (What I say of the lung, of course, applies equally well to some other organs; but the lung is, perhaps, the organ with reference to which one most frequently wishes to consider this point.)

The physical condition, of course, may alter. A normal lung may be roughly described as a spongy structure; as the result of disease it may be solidified: it may become perfectly solid; the air may be entirely extruded from it either by the filling up of the air-cells with pneumonic products, or by compression of the whole lung so as to empty the air-cells of air. Again, it may be excavated—that is to say, it may be hollowed out into larger continuous cavities than the small minute cavities which we know as the air-vesicles. Then, again, there may be fluid collecting in the bronchial tubes. Thus the physical condition may in all those ways be altered from the condition in health. These are the physical conditions of the lung, which the “physical signs” give us the opportunity of ascertaining; and we must constantly bear in mind that that is what we find out by physical signs, and that the presence of a particular physical sign does not necessarily indicate the existence of a particular disease.

With regard to solidification. In a certain stage of phthisis, the so-called second stage, one gets solidification of the lung; in the second and third stages of pneumonia also we get solidification of the lung; therefore if we have any physical signs which indicate the lung is solid we do not from this alone know whether we have phthisis or

pneumonia; and that is an important fact to remember. How, then, are these diseases distinguished? They are distinguished partly by the *locality*. That is a matter of natural history. From the nature of the disease pneumonia commonly affects the lower lobe of the lung; phthisis more commonly begins in the upper part of the lung, at the apex. I do not know whether physicians and pathologists have yet got a solution of the question why this is so; we have at present to accept it as a matter of experience. The *history* of a case of phthisis and that of a case of pneumonia are commonly different. Pneumonia is mostly an acute disease; phthisis is commonly a disease of somewhat longer standing. Then there are *associated conditions* that are different in phthisis and pneumonia. In pneumonia you get commonly an acute illness, an intense high fever, flushed face, bright eye, a great deal of active dyspnoea—the respirations being as high as 40, 50, or 60, as in the case of a child in the ward just now; whereas in phthisis you get a certain amount of dyspnoea, but not quite the same extent of dyspnoea and active constitutional disturbance as in a case of very acute pneumonia.

The importance of these points is this, that if pneumonia occurs at the apex mistakes may and do occur. You constantly see them. It easily happens if you simply go up to a patient who is ill and find out that he has signs of consolidation at the apex—dullness, bronchial breathing, and bronchophony—that you will take the case to be one of phthisis. You do not inquire minutely into the history of the case. You recognize the consolidation, and you are carried away by the fact that the locality is that of phthisis and not that common in pneumonia. On the other hand, if you get a phthisis of short history—you may get so-called tubercular pneumonia in which the history is very short—you may be led to consider that you have a case of pneumonia. The physical signs only tell you of consolidation of the lung, and from the natural history of phthisis and pneumonia you have to make up your mind which is the most likely, or which is certainly present under the circumstances.

I remember a case—not under my own observation, but much talked of at the time—which was admitted into the ward with severe pulmonary symptoms. There was cracked-pot sound under one clavicle, the patient was suffering severely from dyspnoea, and the temperature was high. I cannot

give you all the details of it ; I am speaking from memory. At any rate the clinical assistants, who perhaps ought to have been more cautious, were satisfied the case was one of phthisis. The physician, who was habitually by experience perhaps more cautious, heard something of the history, learned it was of very short duration, and was quite content to believe the patient had acute pneumonia, in which, as we know, there may be a very tympanitic, or even a cracked-pot sound. I believe the clinical assistants were a little obstinate in the matter, and were not at all willing to give up their diagnosis ; but the patient unfortunately died, and there was no doubt it was an acute pneumonia.

That is only an illustration where the physical sign or signs by themselves were entirely misleading, simply because they were not fairly balanced and considered in relation to the history and other conditions.

I remember another case which bears on that point. A year and a half or two years ago, a woman came into my ward very ill indeed, and looking emaciated, and as if she had been ill for a long time. She had physical signs precisely like those of a cavity at the left base. Undoubtedly, basal cavities are very rare. I was perfectly alive to that fact, and I was not at all anxious to make a diagnosis of basal phthisis. Still the history, so far as we could get it, that is to say that she had been ill for some weeks, that she was emaciated, and had not the characteristic general symptoms of acute pneumonia,—made me think, in spite of my unwillingness, that it was a case of basal phthisis. For a week she continued very ill, and my opinion seemed to be confirmed ; but after that she gradually improved, the physical signs cleared up, and within five weeks she left the hospital well. There again the physical signs helped to mislead one ; and one was also deceived by a little difference in the general conditions of the patient, and by the impression that she had had her pulmonary symptoms longer than perhaps she had.

You must not be misled by occasionally seeing your teachers or any physician who is your senior arrive very rapidly at a diagnosis apparently by physical signs. You may, perhaps, see him come to a patient, hear a fact or two, as that the man has been ill for so many days or a week or two, and has pain in his side or cough, then he auscultates and rapidly comes to a conclusion the man

has this or that disease,—pneumonia, phthisis, or whatever it may be ; and since he has expressed his opinion immediately after he has auscultated you may think he has formed it in consequence of the auscultation. But you must remember that in an examination of that kind the physician has always before him the patient and a fact or two of the history, and it is often just the appearance of the patient that gives the clue to the condition, for instance, as between phthisis and pneumonia. The phthisical patient who has reached a well-marked stage of his disease is thin, sallow, and anæmic ; whereas the patient with pneumonia is not wasted, as he has been only a few days ill ; he has a flushed face, and generally presents the appearance of a person previously well, taken suddenly ill with acute disease. So with regard to a number of other points, the trained eye of the physician enables him to detect them, while those who are beginners may not as yet have acquired that education.

There are other instructive instances of which you will meet the counterparts several times in the course of your career here. One I shall now take is that of broncho-pneumonia and early phthisis. Here it is not so much a question of the physical signs of consolidation as of that physical sign which we know of under the name of râles or crepitations or cracklings. You know that frequently a very early sign of phthisis is a more or less sharp râle or sometimes only a dull râle, but a râle of some kind in the situation of the commencing disease. Broncho-pneumonia of children is frequently indicated solely by the same physical sign. There may be no dulness, and no bronchial breathing, but over several separate patches of lung you hear râles.

In the clinical ward, a year or two ago, there occurred the case of a girl who came in with a history of five or six weeks' illness, with abundant râles over one chest. She was thin, somewhat cyanosed, her lips tending to blue, and her cheeks bluish. She was expectorating a quantity of pus. Of course, advanced pathologists or clinicians would say, Where was the microscope, and where were the bacilli ? I believe that we did not examine for bacilli ; but you must remember that bacilli are not always found when the disease is phthisis, and, therefore, they are not an absolute test. However, I am dealing with physical signs, and not with the question of bacilli.

Now, the duration of the illness, the presence of

râles chiefly, if not entirely, over one lung, a temperature oscillating up and down (very characteristic of tubercular disease), the emaciation and the cyanosis suggested the possibility—I do not say the certainty—that she might have tubercular disease. I would here say, with regard to the diagnosis of phthisis, that you ought not, except under special circumstances, to come to a hasty diagnosis of phthisis on a short illness. The diagnosis of phthisis is all the more certain if you have a sufficiently long history on which to base your opinion. The shorter the history the safer you are in being cautious of diagnosing phthisis. I think five or six weeks was the duration of this girl's illness when she came in. For three or four weeks there was little difference or improvement in her condition, and, as time went on, we were more and more inclined to believe she had phthisis. After that, however, she began to improve; and when the illness had lasted somewhat over three months, the temperature became normal, the râles cleared up, and she went out well.

That, I think, is a very important fact to remember, that, perhaps more often in children than in adults (this patient was about 14), an oscillating temperature, dyspnoea, and other evidences of implication of the lung, together with râles over a greater or less extent of the lung, may leave you in entire doubt as to whether the patient is suffering from acute tuberculosis of the lung or from broncho-pneumonia. The longer the duration the more inclined you are to look upon the case as phthisical. Certainly, with a short duration you must not be in a hurry to diagnose phthisis. There is very little, except with regard to localization of the physical signs, that will enable you to draw a sharp distinction between the physical signs in the two cases. If the physical signs are scattered about—two or three patches in one lung, another in another, and so on—it is more likely to be broncho-pneumonia; if there is a grouping together of physical signs—one lung more or less affected and the other but little—then it may be either.

(To be concluded).

Antiseptic Compress for Carbuncles:—

R	Hydr. Salicyl.	...	gr.iss to gr.v
	Acid. Salicyl.	...	gr.xv to gr.xxx
	Sp. Vini	...	3iij to 3j
	Aq. Destil.	...	3iij

M. Ft. lotio. Signa: For external application.

A LECTURE

ON

THE FORMATION OF GRAVEL AND STONE IN SPECIAL REFERENCE TO THEIR PREVENTION AND TREATMENT.

Delivered at Cleveland Street Sick Asylum, in connection with the London Post-Graduate Course, Nov. 23, 1893.

By REGINALD HARRISON, F.R.C.S.,

Surgeon to St. Peter's Hospital.

As a part of the London Post-Graduate Course of Instruction, I purpose drawing your attention to-day to some practical points connected with the prevention and treatment of gravel and stone.

In entering upon a subject of this kind it is hardly requisite to lay stress on the importance of possessing some acquaintance with the primary processes involved, and the circumstances under which such formations take place, otherwise our preventive and curative endeavours must necessarily be, to some extent, of an empirical and tentative nature. If we have no knowledge of the mode in which a stone is built up or initiated in the human body, how is it possible that we can so interfere as to render the concurrence of events, essential to this end, inoperative? It was the absence of information of this kind which led the British Parliament, in 1739, to purchase at a considerable cost the recipe of a pretended process for dissolving calculi, and such-like concretions, which shortly afterwards proved itself to be useless.

I propose to consider the subject before us under three headings. In the first place, I will as briefly as possible refer to certain views and experiments, that are recognized as explanatory of the mode in which calculi are put together within the human body. Secondly, the circumstances which seem to favour the formation of gravel and stone as we may observe them in practice as medical men; and, thirdly, the general principles of treatment which appear applicable in their prevention or removal.

For much that we know of the microscopic characters of calculi, their early history, and the precise mode in which the fine inorganic particles, naturally or casually, existing in the urine are drawn together with the same symmetry and design as may be observed in shell formations, we are largely in-

debted to the labours and investigations of the late Mr. George Rainey,* Dr. Vandyke Carter,† and Dr. W. M. Ord.‡ I can only indicate a few points gathered from these works, which seem to be based upon such facts as the practical physician or surgeon may accept and adopt. Each book is deserving of most careful study in connection with this subject. The chief and leading feature in Mr. Rainey's work is the demonstration of a process or force to which he gave the name of molecular coalescence, under whose influence, and subject to certain definite conditions, inorganic particles are drawn together and consolidated into a globular form. This he demonstrated with Carbonate of Lime, and some viscid vegetable or animal substance, such as gum or albumen, and succeeded in making solid masses which he refers to as "artificial calculi." These experiments you will find precisely described under the title of "process for making artificial calculi," and may be readily repeated.

Under the directions given, and by the aid of the microscope, the minute inorganic particles may be seen to arrange themselves as shown in the drawings in Mr. Rainey's work, which I have had reproduced for your inspection on an enlarged scale. Fig. 1 (*a*) shows the somewhat blurred appearance presented by the inorganic particles as they first lie on the field of the microscope in the viscid material or colloid in which they are suspended. In the course of a few hours minute spherules may be observed, as shown in (*b*). This is effected without any possible assistance from the application of external force or pressure, by the mutual attraction of the two globules, and result in the perfect coalescence of the Carbonate of Lime into small spheres "perfectly transparent, of a hardness nearly equal to that of glass, and giving the sensation when rubbed forcibly by the finger on a smooth, hard surface of small glass beads."

In fig. 1 (*c*, *d*, and *e*) further stages in the process of coalescence may be studied, and the appearance on the field is shown of dumbbell-like bodies and elliptical particles of different excentricity. Fig. 2 (*a* and *b*) marks other stages in

the process of coalescence; whilst in figs. 3 and 4 we have the concreted masses gradually assuming the secondary or globular form. After demonstrating in what manner the spherical figure is first given to the component globules, and how these become incorporated in those of the larger size, so as to result "in the production of the same globular form and of the same structure as those of which they are made up," Mr. Rainey proceeds to consider "molecular disintegration." This he speaks of as "the complete separation and dispersion of the molecules of certain compound calculi, leading to their total disappearance."

To appreciate this retrogression or disintegrating process, we must recognize, as Mr. Rainey points out, that the molecules of which the stone consists are kept together by the action of forces which he speaks of as "gravity and the attraction of tenacity;" forces which can only be operative under certain conditions of the solid relative to the fluid medium or colloid. "When the spheres," as Dr. Ord puts it, "thus formed are plunged into new solutions of different specific gravity, most effectively of greater specific gravity, the spheres lose their concentric arrangement, and break up into radial lines, and subsequently into molecules," reverting, as Vandyke Carter describes, "to the state of granular debris." Mr. Rainey illustrates the process of molecular disintegration thus referred to, by fig. 4 (*a*). Here, then, we have, accurately and fully described, two processes—one by which a mass, such as a urinary calculus is built up, and another by whose operation the stone can be resolved into its previous elements.

It is important we should recognize that the process of molecular coalescence is quite distinct from those formative acts which are the result of crystallization, or are brought about by the mere aggregation, into a more or less solid mass, of certain urinary salts by the agency of a gum or cement. An examination of calculi is quite sufficient to indicate that by neither of these ways can their formation be explained. In order that you may understand my meaning the more clearly, I place before you for comparison three masses of stony hardness, (1) formed by crystallization, (2) by a cementing process, and (3) by molecular coalescence, as we see in these sections of calculi. Nor must we regard molecular coalescence in the light of what physiologically we should speak of as a vital action in contra-distinction to a physical one. Rainey observes, "there seems to be no reason why

* On the mode of formation of shells of animals, of bone, and of several other structures, by a process of molecular coalescence, demonstrable in certain artificially formed products. Churchill, London, 1858.

† The microscopic structure and formation of urinary calculi. Churchill, London, 1873.

‡ On the influence of colloids upon crystalline form and cohesion, with observations on the structure and mode of formation of urinary and other calculi. Stanford, London, 1879.

this explanation of the formation of these urinary calculi by molecular coalescence should not be regarded as the correct one. Vitality has not been named as having had any share in the formation of these natural products. Nor, as it has been demonstrated that bodies in all respects analogous to these calculi can be formed without any possible vital interference, does there appear to be any necessity to attribute any part of the formation of these urinary deposits to the influence of vital force, or to cell development." It is important to recognize this, otherwise it might be assumed that because stony concretions not unfrequently take place in various parts of an organized body, they are necessarily dependent for their production upon some vitalizing agency.

I will now pass on to the second part of the subject and refer to some of the circumstances which seem to favour the formation of gravel and stone, as we observe them in practice. I shall assume that you accept the view offered on the authority of those mentioned, as to the probability of urinary calculi being formed as I have represented. It may, however, occur to you when we consider the rarity of stone relative to the mass of the population, in conjunction with the fact that the urine of all healthy men, women, and children contains certain elements necessary to the formation of stone if placed under favourable conditions, a position is presented which would seem at variance with the theory of molecular coalescence. Surely, one would say, as most urines contain crystals and colloids, or, in other words, salts and an animal basis, if there is any truth in molecular coalescence being the aggregating force, stone at all ages and under all circumstances must be far more frequent than is really the case. Such a criticism as this would seem to apply with still greater force to those instances, by no means uncommon, where persons with enlarged prostates excrete considerable quantities of mucus with their urine but do not necessarily suffer from stone. This, however, would be an inference drawn from an assumption that the mere casual concurrence of crystals and colloid in the shape of mucus or albumen was all that sufficed to determine this end. If, however, we refer to Mr. Rainey's original papers we shall find that to bring about the molecular coalescence of crystal-line particles, design and great attention to details are required. In the first place we may note, in passing, that the title of one of his earliest papers

is "*Precise* directions for the making of Artificial Calculi" ("Trans. Microscopical Society of London," vol. vi., 1851), thereby implying that the conditions are by no means variable or casual. In a given urine you might have, for instance, uric acid or oxalate crystals and mucus or albumen present in abundance, and yet other conditions absent which are equally essential to the completion of molecular coalescence. This is evident in following out such directions as these: "the process of artificial stone-making consists in introducing into a two-ounce phial, about three inches in height, with a mouth about one inch and a quarter in width, half an ounce, by measure, of a solution of Gum Arabic saturated with Carbonate of Potash. The specific gravity of the compound solution should be 1.4068, when one ounce will weigh 672 grains. This solution must be perfectly clear; all the Carbonate of Lime which had been formed by the decomposition of the Malate of Lime contained in the gum, and also all the triple phosphate set free by the alkali, must have been allowed completely to subside," and so on.

When we, therefore, consider the difficulties in the way of securing in an organized body such uniform conditions as are here referred to, we can understand the exceptional nature of calculous complaints, relative to the population, without setting aside, on the ground I have indicated, the explanation of the process of stone-making by molecular coalescence as being inapplicable to the human species. If regard must be paid, for instance, to the temperature, reaction and specific gravity of the urine, at the same time knowing how varying circumstances such as heat and cold, diet, exercise, and the like, may easily influence these in the one direction or the other, it becomes apparent that though happening and effective, the concurrence of the conditions within the urinary apparatus necessary for molecular coalescence, must be, and fortunately is, not very common. Nor is there anything incompatible with the theory I am advocating, that the distribution of calculus throughout the world, or in a limited country like our own, is so variable, as Dr. Plowright shows by figures and a map.* When the attending circumstances are such as to add one or more conditions favourable to the process of molecular coalescence to those already existing or to intensify others,

* On the cause and distribution of calculous disease. Illustrated with coloured maps, showing the distribution of calculus and rainfall in England and Wales. Printed by Jackson Gaskill, 16, Paternoster Square, 1886.

then the probabilities of stone or gravel forming are proportionately increased, and a greater number of cases coming under this denomination may naturally be expected. In this way we may explain the preponderance of stone cases in certain parts, which are spoken of as stone districts..

Having thus, I venture to think, fairly met an objection that might be raised in a general manner to the doctrine of molecular coalescence and disintegration, in its application to the formation and dissolution of urinary calculi, I will proceed to consider certain circumstances which may be regarded as in some way contributory towards the production of such concretions. I would, however, lay stress on what appears tolerably certain, that there is not an individual in fair health who is incapable of forming a stone in a reasonable time, provided that the other conditions favourable to concretion are present. If, for instance, a man, who, in the course of his normal health, is in the habit of excreting ten grains of lithic acid daily, was only to void half this quantity with his urine, and concrete the remainder, it is obvious he might form a stone of this material alone, weighing not less than half an ounce in something like forty-eight days.

That this aspect of the stone question has been somewhat under-estimated, and abnormal contributory products searched for in explanation rather than contributory conditions relative to normal constituents of the urine, seems obvious. If the urine of persons residing in what are generally spoken of as stone districts, such as the eastern counties of this country, and in those parts of India where this disease is very prevalent, materially differed from what may be regarded as a normal standard, there cannot be a doubt, considering the keenness with which analytical chemistry has been applied to this excretion, we should long ago have been acquainted with such distinguishing conditions. But this is not so.

Let us apply these observations to some well-recognized facts connected with the history of stone, and endeavour to see how far they corroborate or are at variance with them. Amongst the most frequent victims of stone in the bladder, we must include male children under or about 12 years of age. That females of a corresponding age are much less liable, owing to the different construction of the parts involved serves as another illustration how mechanical conditions may alone favour the growth of a stone. That the frequency of stone in these young subjects is entirely due not

to chemical alterations in the state of the urine, but to diathetic influences affecting the urinary apparatus itself, seems tolerably evident. Mr. Cadge says, "the prevalence of stone amongst the children of the poor is largely due to the impossibility of their obtaining a proper and sufficient supply of sound milk." And for this in his own district where calculous disorders are so rife, a drinking-water is substituted, "the only peculiarity of which is its excessive hardness." That the use of water of this nature tends to largely increase the amount of mucus naturally excreted in conjunction with the urine, is an observation which I have frequently substantiated. In this way I believe a considerable addition may be made to the colloid or mucoid material, other conditions being favourable, which plays a not unimportant part in connection with the process of molecular coalescence. I have made experiments which have shown that the amount of urinary mucus precipitated is importantly influenced by the character of the fluids that are consumed.

In regard to the action of mucus as compared with albumen as a colloid, Dr. Plowright makes an important remark, on the authority of Dr. Ord, "that whatever tends to keep the colloids diffused will oppose the formation of calculus, for while mucus equally diffused does little more than round the edges of the crystals, precipitated it at once becomes the bed of spheres and agglomerations of spheres." Thus is explained the comparative rarity of calculous affections in persons suffering from Bright's disease of the kidneys—the albuminous colloid being here evenly diffused.

(To be concluded.)

TWO LECTURES ON THE USE OF ANTISEPTICS IN MIDWIFERY.

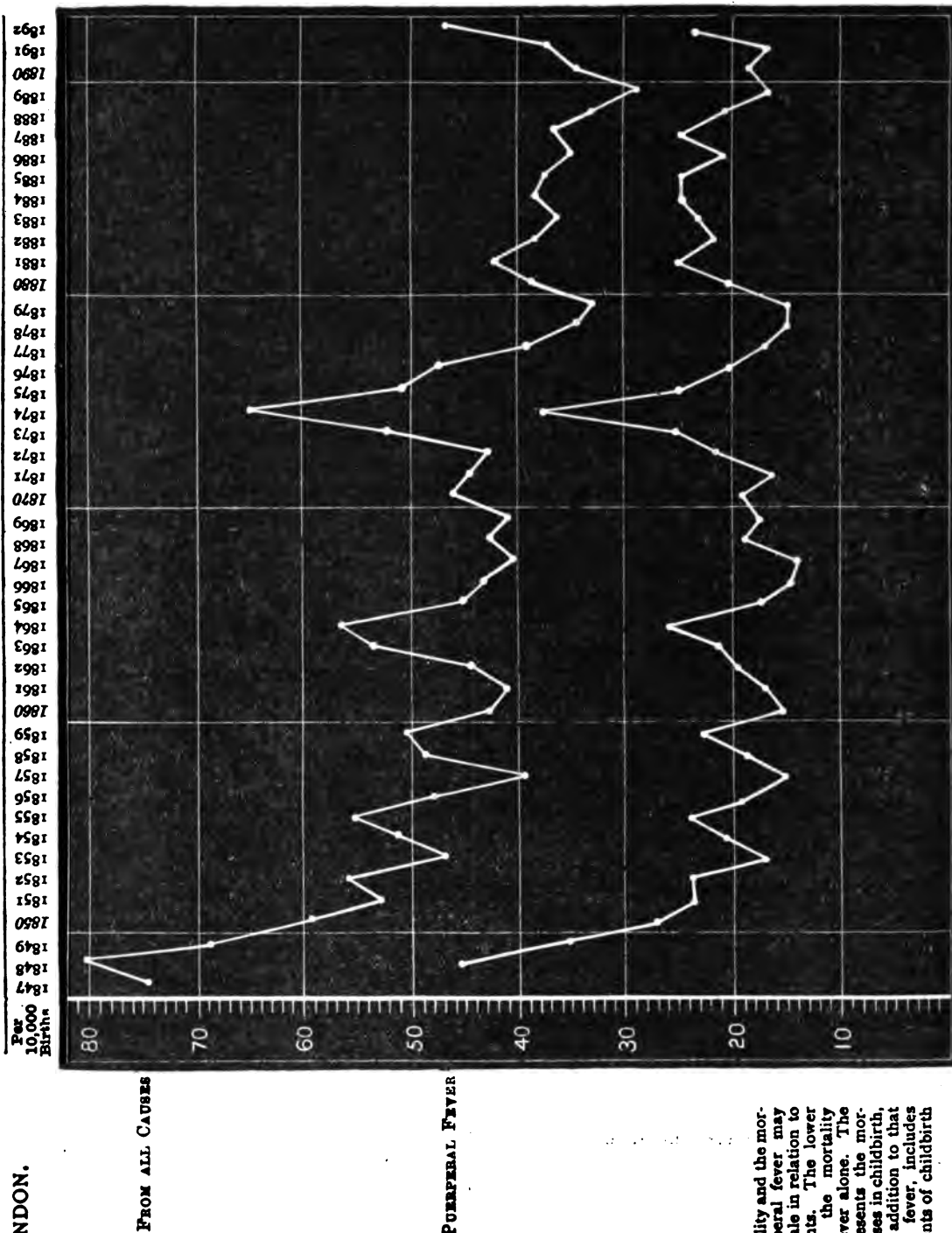
Delivered at the Middlesex Hospital, October 10, 1893,
By ROBERT BOXALL,
M.D. Cantab., M.R.O.P. Lond.,
Assistant Obstetric Physician to, and Lecturer on Practical
Midwifery at, the Middlesex Hospital.

(Concluded from p. 16.)

LECTURE II.

GENTLEMEN,—Permit me first to redeem my promise to show you the mortality charts to which I referred in the last lecture. These charts—one for London, the other for the provinces—give the

CHART I.

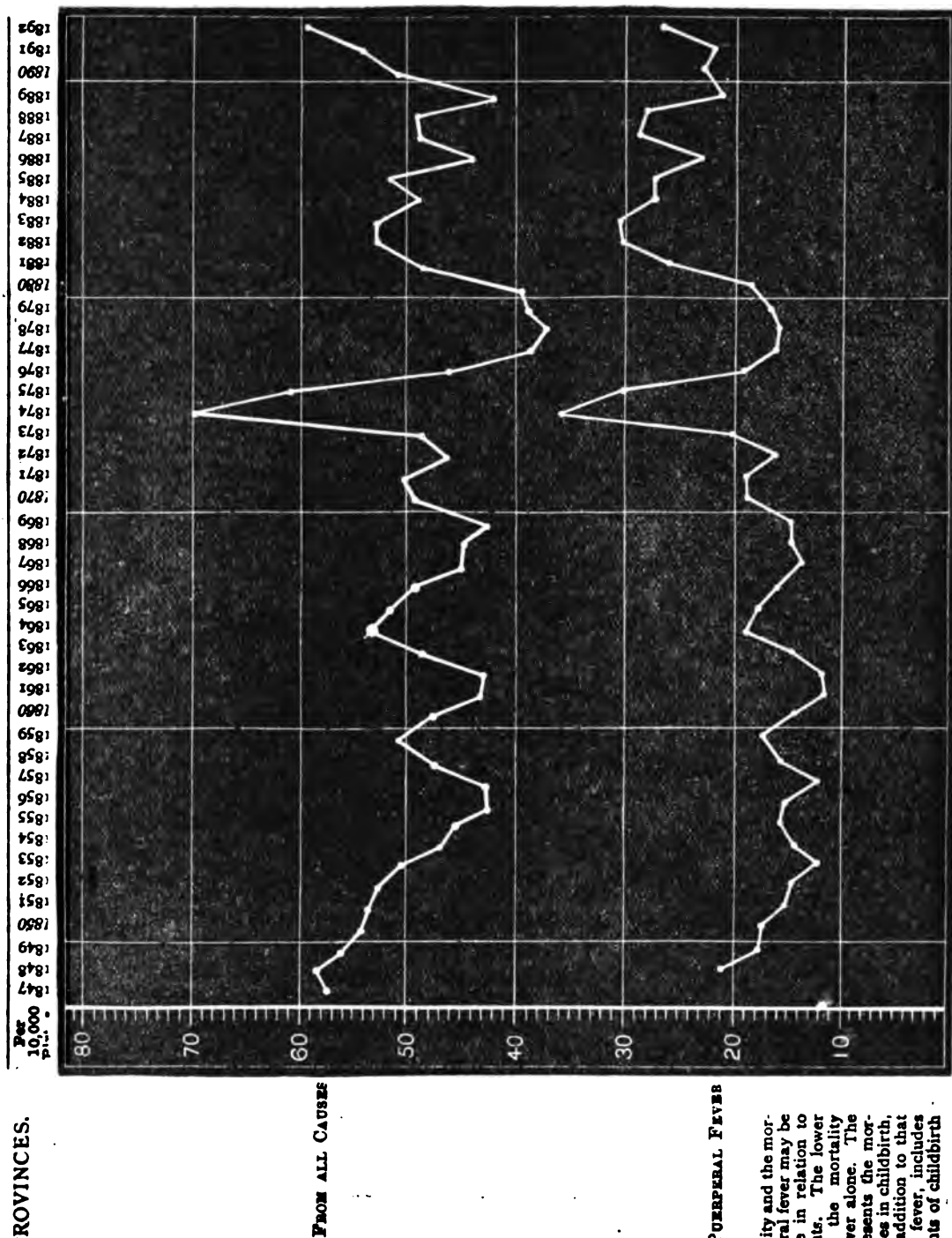
DEATH-RATE OF CHILDBIRTH
IN LONDON.

EXPLANATION—

The total mortality and the mortality from puerperal fever may be read on the scale in relation to 10,000 confinements. The lower curve represents the mortality from puerperal fever alone. The upper curve represents the mortality from all causes in childbirth, and therefore, in addition to that due to puerperal fever, includes that due to accidents of childbirth also.

DEATH-RATE OF CHILDBIRTH IN THE PROVINCES.

CHART II.



EXPLANATION—
The total mortality and the mortality from puerperal fever may be read on the scale in relation to 10,000 confinements. The lower curve represents the mortality from puerperal fever alone. The upper curve represents the mortality from all causes in childbirth, and therefore, in addition to that due to puerperal fever, includes that due to accidents of childbirth also.

death-rate of childbirth, calculated for 10,000 confinements, from the returns of the Registrar-General. Under the separate heads of puerperal fever and of accidents of childbirth, these diagrams give forcible expression to the amount of fatal illness which is directly associated with the process of parturition. No further words of mine are necessary to bring this fact home to you.

In the previous lecture, I merely made mention of antiseptic solutions in a general way. In the present, it remains to consider the choice of a suitable antiseptic, and the preparation of solutions for use.

THE CHEMICAL INCOMPATIBILITY OF ANTISEPTIC AGENTS.

In deciding on the antiseptic for use in any individual case, whether it be a surgical or an obstetric case, it is advisable that as far as possible

to which the application is made, but possibly also on the body generally as the result of absorption. The borderland between safety and success is, in many instances, a very narrow one. The necessity of maintaining a standard strength of the solution is, therefore, important.

I must refer to my original communication* for particulars; it is sufficient for the present purpose to state that, taking Corrosive Sublimate, Carbolic Acid, Iodine, Salicylic Acid, and Condyl's Fluid as examples, I showed that many instances occurred in which, when brought into contact with one another, or with certain lubricants, chemical action took place; and I here reproduce the annexed table for purposes of reference.

The crosses in the table, which occur at certain intersecting points of the horizontal and vertical columns, indicate that interaction takes place between the agents which stand at the head of

	1. Sublimate.	2. Carbolic.	3. Iodine.	4. Salicylic.	5. Condyl.	6. Olive Oil.	7. Vaseline.	8. Glycerine.	9. Soap.
1. Corrosive Sublimate Solution (Bichloride of Mercury).	—	—	×	—	—	—	—	—	×
2. Carbolic Solution (Phenol)	—	—	×	—	×	×	—	—	—
3. Iodine Solution (Iodine and Iodide of Potassium) ...	×	×	—	—	—	—	—	—	×
4. Salicylic Solution (Salicylic Acid)	—	—	—	—	×	—	—	—	×
5. Condyl's Fluid (Permanganate of Potassium)	—	×	—	×	—	×	—	×	×

NOTE.—Wherever a × occurs, interaction takes place between the two substances in the corresponding horizontal and vertical columns.

the same antiseptic agent should be used for all purposes. For if more than one be employed there is always a danger, unless special care be exercised in their selection, that they will interact chemically, and that one will thereby counteract the antiseptic power of the other. All these antiseptic agents, as I some years ago pointed out, are chemical bodies possessing definite chemical properties, which the mere fact of being antiseptic does not prevent them from exercising. The possibility of reducing the strength of the solution, or of altering its nature, through the chemical incompatibility of the materials employed as antiseptics, has an important practical bearing. For, if the solution be too attenuated, the object in view will fail in its accomplishment, and, if too concentrated, considerable damage will in many cases be wrought, not only locally on the tissues

these columns—as follows—Corrosive Sublimate and Iodine, Corrosive Sublimate and Soap, Carbolic Acid and Iodine, Carbolic Acid and Condyl's Fluid, Carbolic Acid and Olive Oil, Iodine and Soap, Salicylic Acid and Condyl's Fluid, Salicylic Acid and Soap, Condyl's Fluid and Olive Oil, Condyl's Fluid and Glycerine, and Condyl's Fluid and Soap. I give the above merely as examples, I must ask you to draw on your chemical knowledge for other instances, in dealing with other antiseptics.

In condemning these admixtures as chemically incompatible, I am prepared to admit that in some instances the newly-formed compounds may possibly be powerful antiseptics, though

* "The Chemical Incompatibility of Antiseptic Agents." Brit. Med. Journ., vol. i., 1888, p. 898; and Lancet, vol. ii., 1888, p. 993.

this remains to be proved; and I submit that any observations made with a view to determine this point are practically valueless, unless either these newly-formed compounds be isolated and used in pure solution, or the admixture be made in such proportion that at least no appreciable excess of either one or other of the original antiseptic constituents be suffered to remain in the solutions employed. Take for instance this, which is commonly known as Iodized Phenol, as an example. It is an admixture of Tincture of Iodine and Carbolic Acid. In the presence of water the free Iodine disappears, as is seen at once by the decolorization of the solution. But that it is still capable of acting as an antiseptic, after water has been added, I do not deny, for it contains much more Carbolic Acid than is necessary to destroy all the free Iodine present. But, that it possesses antiseptic powers, superior to the unaltered Carbolic Acid remaining in solution is extremely doubtful.

One of the least commonly recognized but constantly occurring examples of the use of an antiseptic agent in combination with another body, whereby it is rendered inert, was to be found, at any rate until quite recently, in the employment of carbolized oil for various antiseptic purposes. In fact, at one time, as a lubricant supposed to possess antiseptic properties, the employment of carbolized oil was almost general. And yet it has all along been recognized that in cases of carbolic poisoning, olive oil proves one of the best antidotes. Moreover, many years ago, Koch of Berlin showed that anthrax spores are capable of living and developing after having been immersed in carbolized oil (1 in 20) for 4 months. And yet the fallacy survived, and even in the minds of some still survives, that carbolized oil is a reliable antiseptic product. But that this product contains no free Phenol, unless the strength be raised above 1 in 8, is easy of demonstration. The application at the outset of a little chemical knowledge would have settled the point at once, without requiring years of clinical experience to draw attention to the fallacy by slow degrees.

THE COMPARATIVE VALUE AND SPECIAL USE OF VARIOUS ANTISEPTIC AGENTS.

Perchloride of Mercury.—Out of the whole list of antiseptic agents there is none which has proved as efficacious as Perchloride of Mercury or Corrosive Sublimate. Though it possesses certain

drawbacks they are, on the whole, more than counterbalanced by its advantages.

For disinfecting the hands and certain instruments 1 in 1000 solution should be employed, and for the douche given before, and immediately after, labour 1 in 2000.

It may be conveniently prepared (1) from concentrated solution 1 in 50, which corresponds in strength for antiseptic purposes to pure Phenol, and requires dilution to the same extent.

R Hydrarg. Perchlor.... 3j
Acid. Hydrochlor. Dil. ... 3ss
Inf. Rosæ Dulc.
Glycerin. āā q.s.
Aquam ad 3vj

The Concentrated Corrosive Sublimate Solution. Poison. One ounce of this solution added to one pint of water makes 1 in 1000 solution.

Or (2) from powder, which has the advantage of portability.

R Hydrarg. Perchlor. Pulv. ... gr.x
Acid. Tart. Pulv. ... gr.xl
Pulv. Cocci ... gr.j

To make 1 powder. The Corrosive Sublimate Powders. Poison. One of these powders dissolved in one pint of water makes 1 in 1000 solution.

In either case acid is added in order to prevent precipitation of the Mercury in the form of a white deposit, and consequent deterioration of the solution on standing awhile by the slight alkalinity of water, such as that of London. The colouring matter is added in order that the solution, being devoid of smell, may be recognized by the pinkish tint artificially imparted to it, and, if made to a standard colour, the strength of the solution may always be checked by the same means. The addition of a small quantity of Glycerine is necessary to dissolve the Sublimate in the concentrated solution.

Most of the tabloid preparations which have been introduced contain no free acid, and on standing the solutions become milky for the reason above assigned. I cannot, therefore, recommend them. But in the so-called "soloids" of Corrosive Sublimate, manufactured by Burroughs, Wellcome & Co., this objection has been overcome by the addition of pure Chloride of Sodium. One "soloid" dissolved in one pint of water make a solution strength 1 in 1000. A considerable supply of Sublimate in this form can be conveniently and safely carried even in the pocket.

Prepared in accordance with the prescriptions which I have given, solutions of Sublimate will

maintain their strength provided care be exercised to prevent contamination with soap—which throws down the Mercury as an insoluble mercurial soap—or with bright steel or copper, which deposit it in the metallic state. Very little contamination of either kind, in virtue of the attenuated strength of the solution, is sufficient to deprive it completely of antiseptic power. Blood and serous discharges also precipitate Sublimate from solution. Hence, the necessity of cleansing the hands and instruments before immersing them in Sublimate solutions.

From an extensive personal experience I am of opinion that Sublimate solution is far less irritating to the hands than Carbolic solution of corresponding strength, and less irritating to the tissues locally where used as a wash. It exerts a slight astringent action on the parts, which in the early stage of labour would be disadvantageous were it permanent. But the astringent effect so soon passes off that the astringency can scarcely be considered as offering an appreciable drawback to its use.

The sole remaining drawback to the use of Sublimate as an injection, and the most serious, is the possible risk of mercurialism, even when employed only during and immediately after labour. But this risk may be reduced to a minimum by following out the directions given above for administering the douche, so as to prevent retention of the solution; by insuring, as far as possible, the removal of blood clot; by duly obtaining and maintaining retraction of the uterus, and by immediately closing lacerations about the vulva by sutures, so as to diminish the area for absorption. The special object of effecting the removal of blood is to prevent the albumen from first precipitating the Mercury and “fixing” it, as it were, in considerable quantity, as so-called Albuminate of Mercury. This precipitate, after being gradually redissolved by the excess of albumen, is again rendered liable to be absorbed into the system in the form of soluble albuminate.*

Even under these favourable conditions some slight absorption may yet take place; but, by promoting a free watery flow from the kidneys, and above all by obtaining daily evacuation of the bowels by giving salines in small doses two or three times a day, so as to increase the power of elimination of both these organs, little risk of injurious effects need be feared, even when the douche is given repeatedly.

* Vide “The Use of Acidified Corrosive Sublimate as an Antiseptic.” Brit. Med. Jour., vol. i., 1888, p. 295.

Though I must refer to my original communication* on this subject for further particulars, it is well that I should briefly enumerate the symptoms to which the undue absorption and defective elimination of Mercury under such circumstances usually gives rise. They are not so generally recognized as they should be. These symptoms are diarrhoea, with tenesmus and occasionally blood, as well as mucus in the stools, accompanied by abdominal pain. In severe cases the colon, and to a less extent the small bowel, particularly in the region of the cæcum, becomes ulcerated. Let me warn you, if these abdominal symptoms should occur, not to persist in the mercurial douche, and not to check the diarrhoea abruptly by administering Opium. There is often slight albuminuria, soreness of the gums, loosening of the teeth, occasionally vomiting, salivation, a red line at the margin of the gums, and a metallic taste in the mouth; but these are exceptional in comparison with the abdominal symptoms.

You may judge from what I said in the previous lecture that, despite its drawbacks, I have special reason for advocating the use of Sublimate in preference to any other known antiseptic. From the data which I have given it may be estimated that the net gain effected since the substitution of Sublimate for Carbolic Acid and Condy's Fluid in the General Lying-in Hospital, has resulted in the saving of at least thirty maternal lives from puerperal fever, in addition to 700 cases of non-fatal septic illness often leading to permanent disease. Though I am unwilling to ascribe all the benefits to the use of Sublimate, there is ample evidence to show that it has proved an important factor in the elimination of septic influences. This gain, I admit, has been effected at the cost of one fatality, which must, in the main, be attributed to mercurialism. But, considering that the beneficial results have been achieved at so slight a cost, and that, as the result of experience, we now know better than we did how to recognize the symptoms and to obviate the dangers of mercurialism, I have no hesitation in recommending Sublimate as a douche, provided you pay attention to the points which I have already indicated. The use of Sublimate for douching after labour can only be considered safe in the hands of a doctor who is not only aware of its dangers, but who has also learnt

* “The Conditions which favour Mercurialism in Lying-in Women, with Suggestions for its Prevention.” Obst. Trans. vol. xxx., 1888, p. 304.

how to recognize these dangers and the means of preventing them under different conditions. Even as a vaginal wash Sublimate should never be used by a nurse except under the direct supervision of a doctor, and in no case, when for some special reason it is considered necessary to carry the tube into the cavity of the uterus, should it be employed save by the doctor himself. No objection from danger of mercurialism can be urged against the use of Sublimate as a douche before delivery. And for external purposes, such as disinfecting the hands, the use of Sublimate entails absolutely no risk of mercurialism. At the same time I look forward to the day when some equally efficient, but more satisfactory, antiseptic will be found to take its place.

Sulphate of Copper.—In place of Corrosive Sublimate for the douche, Sulphate of Copper has been advocated, more particularly in France, in 1 per cent. solution. It is prescribed by law for the use of midwives, who in France are under State control. I have no personal experience of it in midwifery. The advantages claimed for it are—that it can be recognized by the natural colour of the solution; it is cheap; it is readily obtainable; it is non-poisonous, even if absorbed, and it causes but little local irritation. I doubt whether, save on the single score of colour, it surpasses Perchloride of Mercury in any particular. Certainly, on the score of antiseptic power it is inferior to it. I cannot, therefore, recommend its adoption.

Carbolic Acid.—Carbolic Acid is powerfully antiseptic, but somewhat less so than Perchloride of Mercury.

For disinfecting the hands and instruments 1 in 20 solutions should be employed, and for the douche given immediately before and after labour 1 in 40.

It should be prepared from pure Phenol (which, by the way, is not an acid) by the addition of water. It is a poison, but may be recognized by its powerful smell, which to many ladies is objectionable, and on that account Carbolic Acid does not invariably commend itself. It is not incompatible with soap, nor destroyed by metallic instruments.

But let me warn you that merely washing the hands in water with Carbolic Soap is by no means a satisfactory means of disinfection. A little reflection will show that the amount of antiseptic thus engaged must be infinitesimal. At least a quart of strong Carbolic solution is considered

requisite for disinfecting the hands. This quantity of solution contains one, if not two, ounces of pure Phenol. How many ounces of pure Phenol, think you, are contained in a whole cake of Carbolic Soap? and what portion of a cake of soap, think you, would be used in washing the hands?

In corresponding strength, solutions of Carbolic Acid are more irritating than Sublimate, and are about equally astringent.

Like Sublimate, Phenol is liable to absorption, and to produce injurious effects, usually ushered in by carboloria—indicated by a peculiar smoky tint of the urine which deepens on standing.

Other Antiseptics.—Iodine solution (two teaspoonfuls of Tincture of Iodine, or one teaspoonful of the Liquor Iodi, to one quart of water, also liable to absorption and to produce a train of injurious symptoms—iodism); Sanitas (one part to three of water); Condy's Fluid (one teaspoonful to one pint of water, the colour of weak claret), and Boric Acid in saturated solution, are all inferior from an antiseptic standpoint, and cannot be so readily depended upon to effect the objects which have been detailed above.

Many other bodies are vaunted as powerful antiseptics, and new ones are being constantly brought forth and "puffed," which are, however, incapable of standing the test of experience. Still less can these be recommended. Let me warn you against them.

SUMMARY.

I would advise, therefore, that Sublimate should be invariably used for disinfecting the hands and instruments, except those of bright metal, and bearing in mind the precautions which I have enumerated, that it should also be used for the purpose of douching; and that when, as may sometimes happen, it becomes necessary to provide a substitute for this purpose, Carbolic Acid should be employed in preference to anything else.

The surface of bright metallic instruments should always be thoroughly cleansed mechanically. After this has been done, they should be disinfected by steeping in boiling water, or in 1 in 20 Carbolic solution. From this solution, each instrument should be taken as required for use; to a similar solution, each instrument, after having been deprived of blood and other discharges by rinsing in water, should be returned after use, and then thoroughly dried.

Let me caution you against the fallacy of first

disinfecting your instruments and then placing them where they are liable to become contaminated before they are used. By placing them, for instance, on a chair, the floor, or the bed clothes, and, in fact, by bringing them into contact with anything which has not been rendered aseptic, the advantage of the previous disinfection may be entirely annulled.

Similarly, beware of reinfesting your hands after having disinfected them. For example, do not wipe them in a dirty towel or soiled napkin, thrust them into your pockets, use your handkerchief, or handle the bed clothes, furniture or utensils. But, if you are compelled to do any of these things, disinfect again before proceeding. Imagine for the nonce that everything not specially disinfected is coated with lamp-black, and act accordingly.

I have purposely omitted, hitherto, any special mention of the lubricant, because, my object being to lay stress on what is essential, I have endeavoured to avoid giving undue prominence to what, at the best, can exert but slight antiseptic power. To my mind it is a matter of little importance whether the lubricant be antiseptic as well as aseptic, provided whatever it be used to lubricate be previously rendered aseptic. It is only when other means fail that any appreciable advantage can accrue from combining an antiseptic with the lubricant. Considering the small amount of lubricant which is actually used, its antiseptic effect at best can be but trifling. It is essential, however, that the lubricant should be at least aseptic. I would caution you, therefore, not only against the lard, goose grease, and cold cream frequently provided, but also against Oil, Vaseline, and Glycerine, which has been left uncovered or into which the finger, without previous disinfection, may have been dipped. As an additional precaution, it is therefore an advantage to use a lubricant to which an antiseptic has been added. Mercurialized Glycerine or Vaseline, 1 in 1,000, or Carbolic Glycerine or Vaseline, 1 in 20, may be recommended: but Carbolic Oil, in the strength usually recommended, contains no free Phenol, nor will the oil part with it under ordinary circumstances. It may not even be aseptic. In recommending the addition of an antiseptic to the lubricant I feel compelled to lay stress on the fact that, only in the light of an additional precaution is this antiseptic lubricant to be regarded, lest it be considered sufficient in itself

to insure asepsis, and to take the place of other more efficient measures, and like Carbolic Soap *et hoc genus omne* reduce antiseptics to a farce and bring discredit on the name.

To sum up, then, I would recommend as necessary:

(1) In every case,—that a solution of Sublimate, 1 in 1,000, should be prepared and set apart for disinfecting the hands, etc., during the course of labour; and afterwards, whenever a vaginal examination is made, or operation undertaken; and that, in every case, the vagina should be douched once during the first stage of labour, and again once after the labour is completed, with Sublimate solution, 1 in 2000.

(2) Under certain special conditions arising during the course of labour,—that, if the labour be unduly prolonged, a second douche should be given; and a similar douche should be always given immediately before any operation is undertaken.

(3) Under certain special conditions arising during the lying-in period,—that, if the douche be required for other purposes than for its antiseptic effect, it is always well as an additional precaution to combine some antiseptic, albeit a weak one, *e.g.*, Sanitas, Boric solution, or Iodine, with it, even if no fear of septic infection is apparent; and that, when given ostensibly to destroy septic material in the passages, a strong antiseptic solution should be used. It seems preferable that the disinfection should be thoroughly effected once for all rather than run the risk of having to repeat the process. And, finally, that whenever the douche is employed for this purpose, particular precautions as to the mode of procedure should be observed upon the lines which I have already laid down.

To recapitulate. I have endeavoured to indicate by reference to the persistent mortality from puerperal fever the importance of the subject to which I invite your attention. I have, further, brought within your cognizance the fact that the mortality from puerperal fever comprises but a part of the total risk to which lying-in women are exposed on the score of septic infection alone. There are more than sufficient dangers incidental to delivery apart from this, to warrant the special plea "for all women labouring of child." The after-effects of illness, even when the patient escapes death, is a serious item. Let me remind you that those attacked by this disease are not the old and decrepit,

but women in the prime of life, often the most healthy. On these grounds this disease is a serious calamity from an economic standpoint, serious especially from the point of view of the patient and her family, and serious, also, on the doctor's account, leading occasionally to the ruination of his practice. I have further demonstrated that, serious as it undoubtedly is, the experience of lying-in hospitals, of outside maternities, and of the private practice of individuals, have alike shown that puerperal septicaemia is a disease essentially of a preventible character. In hospital and out of it I have been witness to the advantage gained by the efficient use of antiseptics in cases under my own direct observation; but I am sorry to say that such results as have everywhere been obtained in lying-in hospitals and maternities by the adoption of antiseptic measures in the elimination of septic processes are not, as yet, apparent in obstetric practice generally throughout the country. The natural inference is that no approach towards the general adoption of antiseptic measures has yet been made. That this state of things exists is, on consideration, not a matter for surprise; for but a small proportion of obstetric practice is at present in the hands of those who have been educated in the use of antiseptics in a really efficient manner. Lest I be misunderstood upon this point, let me say at once that I do not consider that the grave mortality which persists ought to be laid entirely to the door of the medical profession, considering that, as far as can be judged, half, or even more than half, the total midwifery practice is in the hands of a class of self-styled midwives, who have but the crudest notions of ordinary cleanliness, let alone aseptic midwifery; and even when a doctor is in attendance a vast amount of mischief may be, and often is, wrought by an ignorant and incompetent nurse.

In the foregoing remarks I have striven to indicate the method of insuring asepsis in everyday practice. The safe conduct of a labour case in the hands of a medical attendant is largely a matter of training and experience in antiseptic measures. Feeling confident, as I do, that in the light of recent experience lying-in wards can, with safety to the patients, be established, if not actually in, at any rate in conjunction with general hospitals, I, for one, shall hail with gladness the day when, owing to the additional facilities for bringing these matters more forcibly to your notice at the bedside than is under

existing conditions practicable, the Middlesex Hospital reverts to the original intention of its founders and reopens a special ward for the reception of labour cases.

Let me add that, in these introductory lectures, I have dealt solely with the value of antiseptics from the standpoint of the mother. The infant to which she gives birth may, however, lay no small claim to participation in these benefits, if for no other reason, at any rate on the score of freedom from ophthalmia—a fertile source of blindness in new-born children. Moreover, the patient's attendants—doctor and nurse—may also lay claim to some participation in the benefits of antiseptics in midwifery practice. Many cases of illness, not to say deaths, have occurred through accidental inoculation of the examining finger, which the adoption of antiseptic measures, intended primarily to benefit the patient, would have obviated.

In conclusion, let me urge upon you to master the matter at the outset of your obstetric career, and to be thorough in your endeavours to efficiently carry out in practice the principles which I have endeavoured to inculcate. For rest assured that he who with unwashed hands proceeds to dip the tips of his fingers in a would-be antiseptic solution, prepared by adding an infinitesimal amount of some antiseptic to an indefinite quantity of water, and anon proceeds to examine his patient, under the mistaken impression that he is thereby carrying out aseptic principles, deceives himself and, at the same time, exposes his patient to no inconsiderable danger.

I need scarcely add that in midwifery practice you should as far as possible avoid infectious cases, post-mortem examinations, dissections, and the handling of pathological specimens. If in constant attendance upon septic or infectious cases it is obviously advisable to hand over your midwifery cases to a brother practitioner. But you may ask what further should be done if, for instance you have been in attendance upon a case of puerperal fever or erysipelas, or performing an autopsy.

Do not go direct from any such focus of infection to a labour case or to a lying-in woman. Let an interval elapse, and if possible take exercise in the open air. Change your clothing, at any rate your cloth clothing. Take a bath—a Carbolic bath or Turkish bath if possible—and let the head be shampooed, for it is most essential that all the parts which have been exposed should be disinfected as well as the hands. Above all, disinfect

your hands before leaving the infectious case. It is absurd to omit this precaution at the outset—to put on gloves, then to disinfect your hands and to put on them the same pair of gloves again.

If these precautions be taken, the same risk of conveying infection may be at once prevented. Mere abstention from practice, as is sometimes advocated, even though extended over some weeks, cannot be relied upon to obviate the risk. Time may possibly serve to diffuse and to dilute, but certainly will not destroy septic material. Antiseptics, provided they be properly used, will at once destroy septic and all other infectious matter. He, therefore, who would shield his patients from danger must not only use antiseptics, but must learn to use them with intelligence and care.

THERAPEUTICAL NOTE.

The Hypodermic Use of Arsenic.—Dr. Moyer, Professor of Medicine at the Rush Medical College, Chicago, writes* enthusiastically as to the hypodermic administration of Arsenic. He writes as follows:—

The hypodermic use of Fowler's solution has been recommended by numerous writers. Hammond† claimed that the dose which could be administered in this way was much greater than could be safely administered by the mouth. He says:—

"I have frequently given as high as 35 drops of Fowler's solution as an initial dose. It is very certain that the stomach would not tolerate this quantity. Again, I have often carried the amount given by the mouth to the utmost bounds of prudence—till the eyes were puffed and vomiting almost incessant—and then have continued the Arsenic in larger doses, by hypodermic injections, with the result of the cessation of all gastric symptoms and the cure of the disorder."

Dr. Frühwald‡ treated twenty-two cases of chorea minor by this method. He says that redness of the skin at the site of the punctures was common, and two or three abscesses formed. The dose was from 5 to 20 drops, and after the use of thymol and deeper injection no further trouble was observed.

Grunnach§ treated one hundred cases of goitre with hypodermic injections of Fowler's solution, 1 part to 4 of water.

Dumont|| treated twenty-six cases of goitre with injections of 10 minims of Fowler's solution.

It has been noted by former experimenters who have employed Arsenic hypodermically that there was a decided tendency toward the development of abscess as well as painful cutaneous inflammation. These writers have invariably employed Fowler's solution, and its irritating effects were attributed by some to a septic condition of the solution and by others to the spirits of lavender used to colour it. Prof. W. S. Haines suggested that the irritating properties of Fowler's solution when injected into the

cellular tissue was probably due to the free arsenious acid usually contained in the preparation as found in the shops, and that these might be obviated by using the Arseniate of Sodium, a stable salt, readily soluble and of definite composition. Dr. Haines recommends that the pure anhydrous salt be used; as ordinarily found in the shops it may contain from four to twelve molecules of water of crystallization. If this be driven off by heating to 300 degrees the resulting salt has a definite composition with the formula Na_2HAsO_4 ; a 1 to 100 solution of this salt contains 53 per cent. of the Arsenic found in Fowler's solution, so, approximately, the dose of such solution should be about twice that of the Liquor Potassii Arsenitis.

Arsenic is one of the peculiar and exceptional remedies that is much less poisonous when exhibited beneath the skin than when given by the mouth. The reason for this is shown by the fact that Arsenic, like several metallic poisons, focuses itself largely about the upper end of the small intestine, stomach and liver, and toxic symptoms, even if the Arsenic is given in large doses under the skin, will not appear largely in those organs. Thrown into the cellular tissue it is at once absorbed into the general circulation and is distributed to all parts of the system, while if given by the mouth it may make the rounds of the portal circulation several times before entering the general circulation. It is this peculiar fact that makes Arsenic and Mercury so much less irritating when given beneath the skin.

The use of hypodermic injections of the Arseniate of Sodium is entirely free from the objections noted in the use of Fowler's solution. I have given more than five hundred injections of strengths varying from 1 to 10 per cent., and have never noted any irritation, tumefaction, or anything approaching an abscess. Indeed, the solution is as free from irritation as is the use of simple water, and any redness or slight ecchymosis following the injection is invariably due to carelessness in manipulating the syringe.

The average dose of Fowler's solution given by the mouth varies from five to fifteen minims; perhaps the most usual dose is seven and a half to nine minims, and I think very few of us write prescriptions for quantities of Arsenic exceeding nine minims in a single dose. Of course, we may give it by the cumulative method, increasing each dose and then decreasing it, reaching nine to twelve minims, but certainly those are rather extreme doses.

The quantity of Arsenic in one grain of Arseniate of Sodium is 53-100 of the quantity found in one grain of Arsenite of Potassium; that is, a 1 per cent. solution of the Arseniate of Sodium should have approximately about twice the dose of a 1 per cent. solution of Arsenite of Potash or Fowler's solution. As a matter of fact, I have exhibited an arsenic equivalent in the Arseniate of Sodium of 40, 50, and 60 minims of Fowler's solution at a single dose, and it is my common practice to give an equivalent dose of 30 minims of Fowler's solution as my initial injection. The highest that I have ever reached is the equal of three-quarters of a grain of the Arsenite of Potash; that is, equivalent to 75 minims of Fowler's solution at a single dose, and this without producing more than the slightest toxic effect.

I think that the method is worthy of more extended trial, and it certainly in my hands has given exceptional results in the treatment of chorea. I have not found it to be of any value in paralysis agitans or cases of neurasthenia in which I employed it; but in chorea it has certainly exceeded any drug I have employed heretofore, and has far exceeded in efficiency and in cutting short the duration of the disease, the employment of the Fowler's solution by the mouth; this I attribute of course to the larger dose given and the fact that the salt is brought immediately in contact by the blood with the nervous system without having first passed the liver.

* *Journal of the American Medical Association.*

† *Nervous Diseases.*

‡ *Fachbücher für Kinderheilkunde*, 1887.

§ *Schmidt's Jahrbücher*, ccc. p. 195.

|| *Corr. Bl. für Sch. Ärzte.*

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A CLINICAL LECTURE ON THE VALUE OF PHYSICAL SIGNS.

Delivered at Guy's Hospital, Nov. 11th, 1893, by
FREDERICK TAYLOR, M.D., F.R.C.P.,
Physician to the Hospital.

(Concluded from p. 84.)

Another very important instance illustrating the relation of physical signs to one another is that of fluid or liquid in the chest. A patient comes to you who does not look very ill, is fairly well nourished, but who has been ill a week or ten days; he is short of breath, and is said to have had pain in the chest. On percussion you find the chest dull on one side, there is a loss of tactile fremitus, and the breath sounds are deficient. You come to the conclusion that the patient has pleuritic effusion, and if you explore with the needle you generally find you are correct. There you might feel that the physical signs determined you to make that diagnosis, and so of course they do finally. But take another instance. A patient came under my care five or six years ago with a history of pain and distress in the epigastric region. For three or four months she had been in a great deal of distress, and she was wasted and ill. Examination revealed a great deal of fulness in the hepatic region; the liver was either enlarged or depressed. Behind there was dulness of the chest on both sides up to very near the spines of the scapulæ; and there were over the same region absence of breath-sounds, absence of tactile vibration, and absence of voice sounds. What was the condition here? Had she double pleuritic effusion? It seemed possible. But would the physical signs tell us positively she had double pleuritic effusion? Certainly not. Here was a case in which everybody, I think, would have hesitated and ought to have hesitated, in pronouncing at once that she had pleuritic effusion on both sides, though I believe there were some gentlemen undergoing some examination at the time who diagnosed pleuritic effusion. Eventually we explored. Out of the left chest we drew a

thick bile-stained grumous fluid; out of the right there came clear watery fluid which was undoubtedly hydatid fluid. On both sides of the chest there were hydatid cysts, one of which, on the right side, was contained within the liver, as was ascertained post mortem. Both these collections of fluid were beneath the diaphragm, and not in the chest. Still, you might say, there was liquid; the only difference was as to its seat, and you cannot be expected to tell whether the fluid is on the one side or the other of the diaphragm. But you must remember this well-grounded fact—that solid malignant disease of the lung will give you physical signs that are in some cases absolutely indistinguishable from those of liquid; and it is a warning we have received from our older teachers more than once, that in patients of a certain age—about 60 and over—you must be cautious in drawing conclusions in favour of liquid from the physical signs alone. I think I am right in saying that at one of our sister hospitals in London there was a case that was believed to be pleuritic effusion, from which two explorations failed to withdraw fluid, but in which on using the knife there was removed some cheesy material, subsequently shown to be part of an enormous mass of sarcoma in the left pleural cavity. And this was in a patient aged only 19.* These are rare instances, but they are to be borne in mind.

The point is that the physical signs of fluid are not to be relied upon even as indicating fluid; that a solid material like cancer may produce precisely the same signs.

Now, why, if that is the case, you may ask, do we succeed in diagnosing pleuritic effusion so often. Well, it is entirely because we have the history of the patient. We have the knowledge that the disease is of only short duration, and that practically it is the only disease present. If you have a patient perfectly well a short time ago, taken with trouble in the chest, sometimes with and sometimes without pain (which is not always present to act as a guide to pleurisy), who is short of breath, and presents the physical signs we have learnt to associate with fluid, then you are very likely indeed to be right; but if the patient has

* Clin. Soc. Trans., vol. xiii., 1880, p. 200.

been ill a long time and has other illnesses, such as Bright's disease or heart disease, or if there is some trouble about the abdomen, something wrong about the liver, or any displacement of the costal margin to one or other side which shows there is something below the diaphragm pressing upwards, you must be careful of coming to a hasty conclusion that pleuritic effusion is present.

As bearing on that question I should like to say a few words with reference to the value of the physical signs of fluid in the chest. These we take to be the common physical signs of fluid—dulness, loss of tactile vibration, loss of voice- and breath-sounds. Of course, with these present, you will, for the most part, be right in diagnosing pleuritic effusion; but it is useful to bear in mind that those physical signs I have just enumerated are all *negative*. They cannot be physical signs of a *positive* condition. What is proved by absence of resonance, of breath-sounds, of voice-sounds, and tactile vibration? Nothing *positively*. If you auscultated over many parts of the body you would get these signs; for instance, over the thigh, or the shoulder, or the liver. It does not prove the presence of fluid. All that these signs prove is that the spongy lung, which ought to be there, is not there as a spongy air-containing organ. If it is there it is solid or compressed, or it is pushed aside and something has got into its place which does not give the same physical signs. Therefore, these four physical signs of fluid upon which we rely very much indeed, are physical signs of the absence of spongy lung. That absence may be brought about by liquid getting into the pleural cavity and compressing the lung; but it may also be brought about by anything in the abdomen pressing upwards and doing the same thing. Dulness and the other physical signs will follow in the latter case, as the lung, being compressed, is no longer spongy but a solid substance; the air does not lie in it, and does not move in it. There are other conditions in the chest which may compress the lung from the front. Pericardial effusion may compress the lung from the front, and give you the signs of pleuritic effusion. A very large aneurysm will sometimes do the same thing.

That there are positive signs of fluid I do not deny. A positive sign of fluid is displacement of the heart to one or other side, a displacement of the liver downwards, bulging of the chest or of the intercostal spaces. Ægophony, some of you would say, is a positive sign, but that is quite doubtful.

We must be clear in our minds as to the exact mode of production of ægophony before we can say that.

Of course, so far as physical signs are concerned, I may say that not only do these various physical signs not prove that fluid is present, but even if you allow that fluid is present they do not prove that there is *pleuritic* effusion, for there may be dropsical serum, pleuritic serum, or pus; all of which will give signs of fluid.

Physical signs, then, are not absolute in value. They do not indicate the disease, but only the physical conditions which result from the disease. This general statement may, perhaps, be qualified to a certain extent in this way. It may be said we do not know the explanation in the fullest detail of some physical signs, such as bronchial breathing, tympanitic resonance, and ægophony. But if we do not know the explanation with certainty, in detail, we, at least, know, with regard to bronchial breathing, that it occurs in conditions of solidified lung with patent bronchial tubes; and it is for acoustic physicists to work out exactly what are the ways in which these conditions give us the result. The same may be said of tympanitic resonance, of which we had a good instance in the case of a child suffering from pneumonia—tympanitic resonance occurring over the solidified lung. You sometimes do not get the ordinary dulness, but a condition of tympanitic resonance; and you have got to learn that tympanitic resonance does not necessarily mean a cavity in the lung.

There are two physical explanations given of this sign; and possibly both of them operate under different circumstances. One is that you percuss through solid lung upon a large bronchus, and get a broncho-tracheal resonance. As this tympanitic resonance, in the instance I have mentioned, occurred under the clavicle and down the inner side of the front of the upper lobe of the lung, it is possible that is the explanation in this case. Another explanation may, perhaps, sometimes be the true one,—that a certain portion of lung near the surface is yet spongy and in its natural condition; that beneath it there is solid lung; that the tension of the septa in the healthy portion of lung is modified by the pressure of the solidified lung, and so the conditions of vibration are altered, and tympanitic resonance is the result.

That may operate in some cases; as, for instance, in a case in which I observed well

marked tympanitic resonance over the back in the infra-spinous region; as it seems unlikely that one could get a well-marked broncho-tracheal resonance there.

With regard to ægophony that is a question of physical signs which almost wants separate discussion; but on the whole I do not think it is a thing we can always rely upon. It more frequently occurs under conditions of pleuritic effusion than any other; but we see conditions of lung independent of fluid, in which ægophony has been heard. I think it can be demonstrated that fluid in the pleura and fluid compression of the lung is not an absolute necessity of ægophony. It much more likely depends on some conditions of the bronchial tubes, which are most often produced by fluid, but are sometimes not.

It may be asked, and very fairly, Are there no physical signs that are pathognomonic of disease? It would, of course, be very convenient if there were;—if we could take hold of a certain sign and say, Here we have phthisis, pneumonia, pleurisy, or whatever it might be. That is practically to ask, Are there no physical conditions of the lung brought about by disease, and differing from health, which can be produced by one disease, and by one disease alone. There are certain physical signs that have been at different times treated of as if they were pathognomonic. One, for instance, is cracked-pot sound; another is the so-called fine crepitation of pneumonia; another is the splash of Hippocratic succussion. Metallic tinkling and pectoriloquy may also be alluded to.

Now take cracked-pot sound. Is it pathognomonic?

Certainly not. I have told you of a case in which it led some eager students into a sad mess. Cracked-pot sound occurs of course in phthisis; but you often get it in pneumonia, and perhaps less commonly but under more exceptional conditions, in relation to Skodaic resonance; that is in that portion of the lung above the pleuritic effusion, which is practically healthy, though under compression by the effusion. You get it very often in children's chests which are perfectly healthy. The elastic chest of a child sometimes responds in its vibrations to percussion in a way that will produce the most marked vibration like that of cracked-pot sounds. If the child is crying it comes out all the better. That is a point worth remembering, otherwise in a child wasted from other conditions, such as from starvation, you might get a cracked-pot sound

and imagine you had something very significant indeed.

Again, take the so-called fine crepitations of pneumonia. I must confess I have never been able to feel sure that in pneumonia I detected something I could never hear under different circumstances. I should say that the finest possible crepitation, like the rustling of tissue paper or silk, you can get in conditions of the lung that are not absolutely pneumonic. You get it as the result of œdema of the lung. The physical condition of œdema of the lung is, no doubt, very like the physical condition of the first stage of pneumonia. But you may get it in an œdema which will not go on to pneumonia; and therefore it is not in itself significant of pneumonia.

Practically the same thing has been called a pseudo-crepitant râle under certain circumstances. You sometimes get the lung hampered in its action by pleuritic effusion or pleurisy without effusion. It does not expand for a few days perhaps; the patient does not breathe fully at that spot, and the air-vesicles are empty and collapsed; possibly even the lung gets a little œdematous. At any rate, some day under these clinical conditions you ask the patient to take a long breath, and there is a fine rustling crepitation; he may produce this sound with two or three subsequent breaths, after which it will disappear, so that if you ask some one else to listen to it he cannot hear it. A few hours later it may have reappeared. It is a condition of partial and temporary collapse of the lung, got over by a forced inspiration, which produces a rustling from the opening out of the somewhat sticky air-vesicles.

These are conditions of fine rustling crepitation, indistinguishable so far as mere sound goes.

About the splash of Hippocratic succussion I will only say that in most cases undoubtedly it is practically diagnostic of pyopneumothorax or hydropneumothorax—air and liquid together in the pleural cavity. Shaking them together you get a splashing sound. The rare condition of diaphragmatic hernia—which, of course, is extremely rare—will give you similar sounds.

Pectoriloquy was at one time undoubtedly regarded as pathognomonic of a phthisical cavity; but that cannot be said to be the case at present. Pectoriloquy, as we understand it now, is constantly present in pneumonic consolidation, as well as in phthisical cavities.

I should have liked to tell you more of the

physical signs of broncho-pneumonia in children, of which we have had several cases. But time will not allow me.

I have only to say further, that if what I have said has brought more prominently before you than ever before the fact that physical signs do not correspond to diseases so closely as you may have thought they did, and if you think that this on the whole is a very puzzling and disheartening fact to have to confess, you must remember that Nature did not arrange diseases for the convenience of students going up for examination, nor for that of physicians and practitioners anxious to make an honest living.

A LECTURE
ON
**THE FORMATION OF GRAVEL
AND STONE IN SPECIAL REFER-
ENCE TO THEIR PREVENTION
AND TREATMENT.**

Delivered at Cleveland Street Sick Asylum, in connection with the London Post-Graduate Course, Nov. 23, 1893,

By **REGINALD HARRISON, F.R.C.S.,**
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(Concluded from p. 87.)

Passing towards the other extreme of life, where stone in males is still more frequent, we shall find a variety of circumstances favourable to the process of molecular coalescence. The frequency of stone at or about fifty years of age, and subsequently, is largely due to the fact that individuals have a tendency to begin to eliminate what I would speak of as inorganic gout products at this period, more through their kidneys than their skin. The urine is constantly highly acid in reaction, and crystalline and amorphous products of uric acid and urates in excessive quantities make their appearance on very slight provocation. I do not mean to assert that because a person passes, either habitually or casually, large quantities of uric acid in his urine, he should necessarily incur the risk of stone; on the contrary, we must all know persons who constantly do so, yet neither suffer from gravel nor calculus. I would,

however, emphasize the point, that, in so doing, the individual concerned is much in the same position as the child who is fed on hard water instead of good milk, and that, by causing an excess of urinary mucus from the channels through which the abnormal urine flows, an additional factor in the process of molecular coalescence is almost imperceptibly provided. This is a point which may be readily tested by allowing different urines to remain quiescent in a glass for some hours for the purpose of estimating the relative amount of precipitated mucus contained. So long, however, as the urine passages remain normal, and the mechanism of micturition natural, the liability to concrete excessive or even natural quantities of uric acid in the adult is small, but as age advances and these conditions become altered by the occurrence of changes involving the neck of the bladder, the tendency to suffer from such formations is considerably increased.

By the growth of the prostate, as shown in the figure, in conjunction with a reduced power of micturition, the bladder and urinary apparatus is apt to undergo changes in form which chiefly affect the outlet of the former. Persons who have been in the habit of voiding naturally their urate excretions either in the form of small renal calculi or crystals, cease to do so, the former becoming trapped in the bladder behind the enlarging prostate, or in some of the numerous depressions in the floor of the viscus, and in this manner the conditions favourable to the process of molecular coalescence, as may be observed in the test tube, are added to. Thus diathetic stones may attain a considerable size without exciting any very obvious indications of their presence. Unlike the stones of early childhood, the latter, by reason of the recesses in which they are partly contained, are not liable to much movement within the interior of the bladder; and stones are known to go on, as it were, growing quietly, until acute symptoms are developed by their sudden dislodgment from the part of the organ to which they may have been almost entirely limited.

In these different ways, and in others that might be mentioned, if time permitted, we can study how the various conditions favourable to molecular coalescence may be provided, and their concurrence casually brought about.

It will be observed that hitherto I have made no reference to the varieties of calculi, so far as

their chemical composition is concerned, or endeavoured to draw any distinction between what I have spoken of as gravel or stone. When I see discussions going on relative to defining these, I am reminded of a response once given I believe in the witness box by a countryman. On being asked to indicate the presumed magnitude of a certain object, replied that "he thought it was about as large as a piece of chalk." Where gravel ends and a stone commences I certainly should not like to attempt to define, the one imperceptibly runs into the other, and so long as the precise weight of that which is removed or naturally escapes, is given for purely scientific purposes, I do not think it necessary to trouble ourselves further. In illustrating the various kinds of stone formation met with in the human species, we can have no better example than is afforded by the examination of the section of almost any specimen of what is known as a compound calculus.

To broadly study the conditions under which a stone is formed, I will take the section of a calculus made up of more than one variety of typical ingredients such as is depicted in the drawing before you. Examination shows that the central portion of the stone consists of a concretion of lithic acid deposited in irregular outline. It will be seen that as the process of stone formation advanced the mass gradually increased in size by the deposition of lithic acid, probably representing a natural, though excessive, product of excretion, until a considerable bulk was attained. Then the margin of the lithic acid, for some reason or other, became extremely uneven, and no doubt excited local inflammation. This was followed by a change in the deposit, in the course of which the rough concretion of lithic acid was covered in by layers of triple phosphate as effectually as if it had been encased in a mould of plaster-of-Paris. In this way the stone was smoothed down by having its inequalities rounded off, inflammation subsided, and the deposition of lithic acid returned and continued until a circumferential band of some depth was again formed. Ultimately the process of lithate formation is again interrupted by the recurrence of inflammation, which was doubtless re-excited within the bladder by the considerable size the stone had now attained, and phosphates were a second time substituted.

In this way we may explain the formation of compound or alternating calculi. The clinical histories of many patients I have observed warrant

the assumption thus drawn from an examination of their calculi as to the probable sequence of these events. As botanists and geologists are enabled to explain much relating to the history and age of vegetables and rocks from an examination of their sections and strata, so the pathologist, by a similar process applied to calculi, can describe many of the clinical circumstances which characterize their development and increase—how at one time they progress unostentatiously by an aggregation of that material which represents a constitutional disorder or a diathesis, whilst at another their appearance and the massing of phosphates upon them speak of intense vesical irritation, alkaline and offensive urine, and such-like local distress; and what appears to be true of lithic acid stones applies to other varieties of calculi, with the exception of the triple phosphate, which seems to be, either independently or relatively to other calculi of which it may form a part, a product of inflammation. The latter conclusion is supported by a reference to those numerous experiments which both men and women are frequently trying upon themselves. If a foreign body is introduced into the bladder and allowed to remain there, inflammation is excited, the urine undergoes decomposition, and phosphates are thrown down and deposited upon whatever may be occupying the interior of the viscus. This observation is of interest, as it cannot be doubted that the process of phosphate formation occurring under these circumstances is really a conservative one, having for its object the encasing of that which is foreign and irritating to the bladder. It will be noted that under all states of inflammation in connection with foreign bodies, of whatever kind, the formation that takes place is invariably phosphatic, this process possessing the power of immediately, though temporarily, suspending any other deposition which may have preceded it, as observed in the case of rough calculi.

Hence we can divide the constituents of the various calculi met with in the human urinary system into two classes:—(1) Those made up for the most part of inorganic or crystalline particles which either usually or casually exist in the urine. These concrete by the intercurrent of conditions favourable to the process of molecular coalescence, quite independent of inflammation, or the provision of a foreign body to act as a nucleus. Of these we may mention the urates and oxalates, which seem to be mutually convertible, and cystine, in addition to other rarer varieties; and (2) Those

made up of inorganic constituents derived from the decomposition of the urine, and produced in the first instance through an inflammatory process, and subsequently welded together by molecular coalescence. This variety has already been illustrated as forming a part of the compound calculus of which a section has been described, and can be produced at will. It consists of triple phosphates, as shown in the specimens before you. This formation is chiefly seen in connection with the presence of certain bodies of a foreign nature which may happen to be introduced into the bladder. Amongst such causes may be mentioned various articles which, having been passed up the urethra, have made their way into the viscus and remained there, at least for a time. I can recall to mind in my own practice, as forming the nucleus of this kind of stone in the bladder, pins, needles, wires, a knitting needle, a slate pencil, a feather, a bulb-headed grass, pieces of catheters and bougies, a whole bougie, and a pencil-case; but taking the experience of others this list might be considerably extended. Further, in the same way, this deposit is provided for encasing other stones that may descend into the bladder from the kidney as well as the particles of calculus which are sometimes left behind after lithotomy when the bladder is much pouched or sacculated; and thus a recurrence of stone may be explained. Nor must we forget that certain masses of flocculent material, which are sometimes seen in the urine of persons suffering from chronic cystitis are just as capable of furnishing a nucleus for a triple phosphatic stone, as a feather, a villous growth, or a rugose bladder.

Let me now offer a few remarks in regard to the third division of my subject on the prevention and treatment of gravel and stone. In the first place, the presence of the former may be generally accepted as a forerunner of the latter, though a stone often forms, particularly in elderly people, without exciting any obvious indications of its presence. In persons in the habit of passing gravel or small concretions from the kidneys, a transition stage when this does not happen, owing to an increase in size of what has to be voided, or an obstruction in the urine passage not permitting their spontaneous escape, may occur. Though ureters more or less permanently dilated by reason of stones frequently passing down them, no doubt give readier exit for the escape of gravelly material from the kidneys, it must not be forgotten that such a

condition of these tubes may allow of calculi descending, which are too large to leave the bladder by the normal method. Hence, if there is a sufficient suspicion that one of these bodies or concretions is retained in the bladder, as may be inferred by an attack of renal spasm, being followed by unusual vesical irritation, or by the urine being stained with blood after exercise, or by an increase in the urinary mucus, the sound should be passed to make sure of the diagnosis. If a stone is felt, it may now be removed without delay or risk by crushing without inconvenience. I have frequently, under these circumstances, washed out of the bladder such miniature stones with an evacuator catheter without using any other instrument. Thus their sure growth is prevented.

As for what I have called diathetic stones, that is to say, calculi primarily found by molecular coalescence and without inflammation, we have striking evidence that errors in diet may furnish, in one way or another, important links necessary for the completion of the process which I have been chiefly occupied in discussing to-day. Take the instance of the absence of milk, as already referred to in the case of children, and its substitution by coarser and unsuitable foods. Here, in place of a fluid nutriment which is generally regarded as unirritating to the urinary organs, others are substituted of an opposite nature, and thus the urinary mucus is considerably increased. I once saw a female child a few months old suffering from hæmaturia whose urine contained a large number of uric acid crystals, which seemed to be the cause of the bleeding. The patient was ordered to be fed by the bottle on equal quantities of milk, barley water and Contrexeville water, under which the symptoms gradually disappeared. Had this happened in a boy, it is not unlikely a calculus in the bladder would have been the result.

In the same way with the uric acid in the urine of those more advanced in age. Both by its amount, in addition to the irritation it excites in the urinary passages, the liability to stone is not inconsiderable, particularly when it is associated with the occasional passage of a calculus from the kidney, and with some obstruction in completely emptying the bladder. Concretion by molecular coalescence under these circumstances may readily occur.

There are several points of importance in instances coming under this category. In the first

place it is desirable to diminish the quantity of uric acid so voided, or if this cannot be done, to substitute a harmless solution for a highly concentrated urine.

In persons of a decidedly gouty habit, a somewhat restricted diet both in eating and drinking is usually followed by benefit, though this should not be pushed too far, as one occasionally meets with persons who seem quite capable of maintaining a full supply of uric acid on a diet of little else than bread and water. The East Indian apparently, from what I am told, in certain districts where the disease is very rife, concretes stone composed of urates and oxalates on the sparest and most frugal of diets. Hence certain individuals whose uric acid diathesis is strongly marked and often hereditary, do better on a somewhat generous diet with a moderate amount of stimulant, such as still moselle, whiskey, or even light port that has been matured in the wood, provided that they are instructed to flush their urinary organs daily with some suitable diluent. For the latter purpose various means are employed, amongst others being the drinking of hot water in considerable quantities on rising in the morning, or by means of some natural springs. For some years past I have been using the Boro-Citrate of Magnesia, taken in the proportion of one teaspoonful of the powder in a tumbler of warm water once or twice a day on an empty stomach. My attention was first called to this preparation by a paper by Dr. Kochler,* of Kosten, who advocated its use in cases of uric acid calculi and gravel. From what I have seen expelled after it has been prescribed, I am disposed to think that it not only induces a person suffering from uric acid in excess either in the form of crystals or kidney calculi, to take a considerable amount of flushing fluid, but that it is capable of modifying or altering some of the conditions upon which the formation of urate stones depends. We should remember that though we may not succeed in preventing a person making and excreting uric acid crystals in excessive and 'damaging quantities, we may take means, which have proved successful, to prevent this formation injuring the mucous lining of the urinary passages, or lodging within them. If you cannot arrest a suspicious-looking individual, you may at all events take the precaution of seeing him safely off your premises. So in some cases the use of

alkalies in moderate quantities, in the form of Citrate of Potash or Vichy water, often does good, when used with this object. Where a morning aperient is required, the Boro-Citrate may be combined with dried Sulphate of Soda in the proportion of two parts of the former to one of the latter. A teaspoonful, more or less, in a tumbler of warm water on rising being the appropriate dose. Apart from its laxative properties this combination acts as a gentle stimulant both to the liver and kidneys. The evidence as to the value of common salt relative to uric acid, which I need hardly say is the most common variety of stone in one form or another, including the oxalates, must not be passed by without notice. On this point Dr. Plowright observes, "(a) that the presence of salt greatly increases the solubility of uric acid; (b) that the consumption of salt by increasing thirst insures a larger amount of fluid passing through the urinary tract, and, therefore, lessens the probability of calculus; (c) that by keeping the colloids equally diffused, salt tends to prevent the crystalline solids of the urine from agglomerating into calculi." The same author finds from experiment that the addition of two per cent. of salt quadrupled the solubility of uric acid. I frequently combine common salt with the Boro-Citrate of Magnesia as already described in the proportion of three parts of the latter to one of the former. Amongst new drugs Piperazin is now being tried extensively as a solvent for uric acid gravel, and in promoting its elimination. Though I have seen some good apparently follow its use, I am not at present prepared to endorse all that is published and extensively circulated in reference to its value. It is, however, worthy of a trial in suitable cases,

As a rule, in the uric acid diathesis, the saccharine forms of diet may be dispensed with advantageously. The modern, rich, artificially and stall-fed beef, which is a product not of hay and grass, but of various condiments and oleaginous compounds in the shape of oil-cake, compares unfavourably relative to the formation of uric acid with those produced by a more natural system of feeding. Hence, mutton, fish, and fowl are, as a rule, to be preferred as staple articles of diet, with a liberal allowance of well-cooked fresh green vegetables.

On the advantage of exercise where there is a tendency to produce uric acid in excess, or to retain it within the urinary apparatus, it is hardly necessary to insist. In the earlier part of adult

* Berlin. Klin. Wochenschrift, November 3rd, 1879.

life the natural drift at the present day is rather in the direction of excess, and there is seldom much difficulty in getting attention paid to this point. As age advances, though the necessity for physical exercise proportionate to the powers and inclinations of the individual is the same, if not an increasing one, circumstances often render it more difficult to obtain. With the old athlete, cricketer, or oarsman, the aimless peregrinations of seniority often lead to this important factor in the daily life of a seeker after the "*mens sana in corpore sano*" being comparatively abandoned, and penalties being thus incurred. This should not be so: and though the pleasures of the hunting field, and those associated with the gun, may be limited to the few, the majority should not forget that in golfing, cycling, riding, and other interesting exercises, much may be done in averting conditions connected with the excretions which, as age advances, not unfrequently disturb the urinary organs in the special mode I have indicated.

That a restricted diet alone does not necessarily reduce the quantity of uric acid excreted in the urine is frequently observed. A medical officer of a large convict establishment, from a long series of careful observations, told me his experience was to the effect that prison food and sedentary cell labour combined did not alter materially the excretion of uric acid, but when associated with regular exercise on the treadmill a marked diminution invariably followed.

I must not omit to refer to those forms of calculi and gravel which are the result of inflammation occurring within the bladder. These are not unfrequently met with in persons suffering from obstruction and enlargement of the prostate, though the latter is seldom alone sufficient to cause this result. As I have already said, something either in the urine, or derived from the mucous membrane, or from an extraneous source which can play the part of a foreign body, though it may be only of a flocculent nature, is required to complete this process. Under such circumstances we have to depend more upon local than general treatment, and by keeping the bladder clean, the urine normal, and the passages as free from obstruction to the flow of urine as possible, lessen the number of contributory circumstances towards this evil. That bacterial life, relative to chronic forms of cystitis, and less directly to the formation of triple phosphatic stone, presents some problems which have yet to be worked out seems probable.

Sir William Roberts* has recently contributed a case which tends to show that the bacterial states associated with the lactic fermentation, as observed in saccharine urine, are inimical to the bacillary life which is a concomitant of the ammoniacal fermentation, associated with the formation of triple phosphatic stones. That the bacteria affecting the urinary organs are different in their nature and susceptibilities there can, I think, be no doubt. This is evident from such investigations as those which formed the basis of a paper by Dr. Noël Halle,† and are readily demonstrable by the microscope. That some bacilli live in the presence of agencies and influences which are fatal to others, is probable, and accounts for the varying results obtained in cystitis by such bactericides as Mercury, Carbolic Acid, Boracic Acid, Quinine, Peppermint, and other means which are employed for this purpose. The destructive agencies applicable in practice to various forms of urinary bacteria is a subject which would occupy more time than this occasion permits of. At a recent meeting, I listened with much interest to an address by Sir Joseph Lister on the present applications of Antiseptic Surgery. He expressed himself much in favour of the use of Carbolic Acid for this purpose. I cannot help concluding from some observations made in practice, that though Carbolic Acid has proved to be the most universally destructive bactericide and potent antiseptic, there are some micro-organisms met with in the urinary apparatus which resist even its powers unless used in a form inapplicable to living tissues.

In connection with the treatment of stone, reference must be briefly made to some attempts that have been proposed to bring this about by solvent agencies. To produce such an action on a stone in the kidney or in the bladder, as is effected, for instance, by dropping water on a lump of sugar is a method which, in principle, has been much sought after, and with some degree of success. Unfortunately we have to bear in mind that the soft textures in contact with the stone are extremely sensitive to the action of certain fluids which might be advantageously employed for this purpose, and therefore our efforts in this direction are considerably limited. That the dissolution of calculi may be effected in this way is not impossible when we consider the spontaneous changes stones sometimes undergo in the body,—changes which are explain-

* Lancet, February 25th, 1893.

† French Surgical Congress, 1892.

able when we consider what Rainey has pointed out and described under the name of molecular disintegration.

It is rather to the stages which precede the actual formation of calculi—stages which are frequently demonstrable—that our efforts are to be mainly directed in aborting this result. When a stone has attained such dimensions as to render its escape from the urinary apparatus by natural efforts impossible, the time has not yet arrived, if it ever does, when we should feel at all disposed to trust to what are called solvent agencies. The results of lithotripsy, or the crushing operation, when applied under such circumstances, are so entirely satisfactory as to leave nothing else to be desired. The introduction of elements of either danger or incompleteness may be taken as bearing some proportion to the size the stone has been allowed to attain, by the length of its stay in the bladder. When a calculus has been permitted to reach considerable dimensions, amounting, I will say, in bulk to that of a hen's egg, or thereabouts, it stands to reason that the process of removing it, though not differing in principle, cannot be as simple and as innocuous as when, about the size of a bean, it was just too large to make its escape by the natural efforts of micturition. Further, the growth of a stone within the bladder, particularly when associated with some natural enlargement of the prostate, often leads to permanent alterations in the shape and function of the organ, which increase the difficulty of removal, and provide conditions favourable to recurrence.

Though we have to meet varying conditions, both of stone and of bladder, by such measures as are likely to give the best results, there can be no doubt that in endeavouring to lay down a principle for our guidance, on the assurance that a stone has formed or is retained within the bladder, that principle strongly points, so far as my experience goes, to a preference for lithotripsy, or crushing, as being, beyond all doubt, the safest, speediest, and most effectual method of dealing with such cases.

For Diphtheria:—

R.	Pot. Iodidi		
	Sodæ Salicyl.	...	āā 3j
	Syr. Simpl.	...	3j
	Aq. Destil.	...	ad 3viii

M. Ft. mist. Signa: A dessertspoonful to be taken every hour.

ON SEBORRHOEA AND ITS CONSEQUENCES.

A Lecture for the London Post-Graduate Course, delivered at the Hospital for Diseases of the Skin, Blackfriars, Nov. 7th, 1893,

By J. F. PAYNE, M.D., F.R.C.P.,

Physician to St. Thomas's Hospital, and to the Hospital for Diseases of the Skin.

GENTLEMEN,—Before beginning the lecture of to-day I wish to show you some patients illustrating the condition of which I have to speak.

The first is a very characteristic example of a certain kind of seborrhœa, of which I shall have more to say presently. The patient is a man, æt. 36. He has, for some years, had a considerable amount of scurf on the head; his hair is getting thin on the top; he has, from time to time, had slight soreness behind the ears, from slight eczema; he has had a good many boils on the neck; he has some scaly spots on the sternal region of the chest, but they are not very noticeable; and he has had similar spots on the back. In the flexures of both arms he has a little eczema, composed of scaly papules, not moist or vesicular. Since last week he has had some patches on the outer aspect of the thigh, which take the form of papular eczema. The noticeable fact in this case is the mode of extension. The seborrhœa began on the head, and lasted a good many years; there was gradual extension in the form of eczema, to the ears; after a further interval, some scaly patches, which might or might not be called eczema, appeared on the sternum and back; next, the eruption on the arms, which, although on the flexor aspect, where eczema is usually moist, is dry, and finally, a similar condition on the thighs.

The second man has also had a rather markedly scurfy head for a long time:—about twelve years, he thinks. There has been some soreness behind and in front of the ears; and there has also been, what is more typical, a scaly inflammatory affection on either side of the nose, near the alæ nasi (a true seborrhœa of the face). He has had boils on the back of the neck. He has also very characteristic discoid circular patches on the sternum which are red and slightly scaly, with an appearance which can scarcely be called that of eczema. But on the arms there is what I believe to be a different eruption, composed of

scattered red diffuse patches covered with thin scales and having the appearance of psoriasis. He has the same also on the legs, and very much in the psoriasis region. This eruption has continued since he was about three years old, and appears to be ordinary psoriasis.

The physician, whose patient he is, has put him down as a case of psoriasis and eczema seborrhœicum. And I think we must agree that he has had a complication of diseases; the seborrhœic affection coming on in addition to psoriasis. Apart from this, we have here the same succession of things as in the other case:—scurfiness of the head for a long time, eczema (not of a very severe kind) about the ears; scaly patches on the face; discoid patches on the chest and back. We may notice that the hair is very dry; it has been falling off a good deal, and looks dusty; and that the head is irritable.

The third case is that of a girl with seborrhœa of the same kind. The head has been scurfy ever since she had ringworm when about 7 years of age. She is now 19. I don't suppose the ringworm, however, has had much to do with the present condition. She has had soreness behind the ears occasionally. She also has characteristic discoid patches on the sternum and all over the back. She, too, has a habit of wearing flannel garments next her skin at night as well as during the day. She has had spots over the face, not diffuse red patches, but pimples and pustules, a sort of acne, particularly on the chin, nose and forehead. The scaly patches on the chest are also complicated with some pustular acne. This is a noticeable case, because this particular kind of seborrhœic affection on the body is very much less common in the female sex than in the male; the chief reason probably being that women do not, as a rule, wear such warm garments next the skin; and in order that this particular affection should occur on the trunk I think it is necessary that the skin should be kept very warm, and it occurs especially if flannel is worn, and the same garments kept on night and day. The general health of all these patients is good.

These three cases are enough, I think, to indicate the characteristics of seborrhœa. Of these characteristics I shall now give you a short outline.

The name seborrhœa cannot be taken as a satisfactory one. I do not understand the word in its etymological sense. Seborrhœa ought to mean an excessive flow of sebum; nevertheless, in

most cases of the affection there is no excess of sebum, the only alteration, if any, of the sebum being in its quality.

Seborrhœa, as commonly understood, has two varieties, *Seborrhœa oleosa* and *Seborrhœa sicca*. Seborrhœa sicca is also called Pityriasis capitis, and sometimes, from its producing baldness, becomes identical with what has been called Alopecia pityrodes. In seborrhœa oleosa there is no doubt an excess of oleaginous secretion, that is, of a secretion which makes the skin of the head appear oily and the hair glossy. This may form crusts, and sometimes, especially in children, very thick yellow masses looking much like eczema or impetigo; but when removed they show under them intact and uninflamed skin.

Of that form I have not an example to-day, and shall not, therefore, speak of it again.

The second form is the commoner, and, although it seems a slight matter, really has considerable practical importance. It is highly probable that we have more than one affection included under this name; but at present we are unable to distinguish them. Seborrhœa sicca, or the dry form, is characterised by profuse desquamation of epidermis on the scalp. The layers of scales are sometimes dry, silvery, and loose; but, on the other hand, they are sometimes yellowish and rather closely adherent, and hence may be inconspicuous. They occur either in discrete patches at certain parts, or else all over the scalp. These scales do not appear to be greasy; nevertheless, when they are examined microscopically, or treated with ether, it is easy to see that they contain a very large amount of fat. According to Pincus, a German dermatologist who investigated this point, two-fifths of their weight may be extracted by ether. I have never myself succeeded in getting a quantity sufficient to weigh. However, that is the amount he states. Besides this scaly scurfy condition, familiarly known as "dandruff," there is also a certain peculiarity of the hairs, namely, they are dry, there is a want of lustre, they look dusty. As time goes on they are very likely to become thin or fine, by which I mean that the individual hairs become finer and smaller. They also fall out a good deal, so as sometimes to produce considerable baldness. With regard to the question of baldness I shall only say now, that in women marked baldness is produced sometimes by this cause alone. In men one feels some doubt as to whether this condition produces much effect or not, because it is usually associated

with the typical or inevitable baldness of middle or advanced life, and the effects of the two are not easily separated. But I think even in men it is an accessory cause of baldness; in women it may undoubtedly be the sole cause. Last year I showed to the class a girl of 18 or 19 who had scattered diffused baldness over a considerable part of the vertex of the head from this cause alone, and when properly treated the hair soon grew again.

Besides that, there is, generally speaking, a good deal of irritation, but not invariably; there is very often a sense of heat, and there may be actual hyperæmia, causing a heat of the skin perceptible by the hand.

Another point which ought to be remembered is this, that when we examine the scurfs and scales in these cases bacteriologically for micro-organisms, we find two things at least; always a great abundance of the *saccharomyces capillitii*, torula, or yeast of the scalp, which is found in nearly everyone's head, so much so, that it is difficult to say it produces any morbid condition; and in addition a considerable number of micrococci are present—sometimes in enormous quantity—so that when the scales are properly prepared they are found thickly dotted over with the microbes. That is, in my estimation, a very important fact.

The general course of the complaint is this. When seen, as very rarely happens, at the beginning, it is found to be, I think, partial. It begins at one part of the scalp, but its tendency is to become very nearly universal.

With regard to the cause of this condition it is impossible to say that any cause is really known. It certainly is not attributable to any defect of health. Some who have it are in perfectly good health, and there is even a popular impression that dandruff of the head is a sign of strength. Sometimes, of course, it does occur in bad health. I am inclined to think that some people who complain of indigestion have perhaps more of it, or that it is worse while they are suffering from gastric disturbances. At all events, they get more itching of the head, and that attracts their attention to the condition. Itching of the scalp may be, I think, certainly connected with dyspepsia; but it is impossible to suppose that is the original cause of the whole complaint.

Again, it is certainly not connected, as acne is, with any particular age. It does not depend upon any particular developmental condition of the skin or its appendages. It is not related specially to

habits, although, probably, wearing hats a great deal and keeping the head hot favours it to a certain extent; but I do not think this can be regarded as producing the affection.

Since it is impossible to account for seborrhœa in any other way it is natural to suppose that it may possibly be due to some specific cause; and the idea, I think, is gaining ground that this condition of the scalp when it is considerable, is probably due to some micro-organism not yet isolated. And certainly there is nothing to contradict this idea. If that were the case it ought to be in some instances contagious. Probably it is so, because in families I have often seen one case succeed another. On the other hand, there are many negative cases where one person in a family having suffered from it for a long time, none of the others have taken it. But considering the way in which people have their hair cut and attended to by hairdressers without any precautions whatever, the opportunities of contagion, if there be such, are obviously very numerous.

Thus, all we can say about the cause at present, is, that it is probably something specific, but as yet unknown.

The most important thing about this affection of the scalp is not the inconvenience which it produces by itself, but the fact that it gives rise to other affections of other parts which though not really serious are unsightly and inconvenient. The scalp affection itself is sometimes slight, sometimes considerable. It is really a matter for the patient's own decision whether the scurf in the head has reached such a degree that it is necessary it should be treated; but the bad cases and the slight cases show imperceptible gradations; and, considering the probability that it will cause falling of the hair, or even actual baldness, dandruff ought always to receive some attention.

The history of the extensions and consequences of seborrhœa is something like what we have traced in our patients to-day.

In the first place it often happens that, when seborrhœa sicca has existed in the scalp for a certain time, a similar affection begins to show itself in other parts. Frequently it is some portion of the face,—the eyebrows and the forehead above the brows, perhaps most commonly, then the sides of the nose, the *alæ nasi*, and the patches of cheek adjacent to the nose. Sometimes also, though not so frequently, it appears on the chin. It also occasionally extends backwards to the nape

of the neck and to the ears. Then, again, it is very often accompanied by a similar scaly affection of the sternal region and of the back, particularly the interscapular region. In these parts there is an eruption which is a good deal like the scaly affection of the head, that is to say, it is scaly and red, showing hyperæmia accompanied by some desquamation. It seems to me better not to say that this condition is eczema.

This eruption over the sternum and back is undoubtedly the same affection as has been described by a great many different names,—lichen circinatus, lichen circumscriptus, etc.,—and which has also been known at this hospital by a popular name not altogether to be recommended as “flannel rash.”

When this affects the body it appears first in the form of a sort of papule apparently connected with an enlarged and inflamed hair follicle or sebaceous gland. This, when scratched, always shows minute spots of hæmorrhage, not exudation as in eczema. From the papule the affection extends peripherally till it forms a scaly disc. As it goes on, very generally the central part heals up, while the peripheral parts go on growing till it forms a ring, and hence the name, lichen circinatus. But you may trace every gradation, from a papule connected with a sebaceous gland or follicle to the complete ring. The central part of the ring is generally of a yellowish or fawn colour, and this discoloration may extend over considerable areas.

This affection, formerly known as lichen circinatus and lichen circumscriptus, is, I think, fairly described as seborrhœa corporis, since, as was first suggested by the American dermatologists, it corresponds to seborrhœa of the scalp, and is almost always accompanied by that condition.

The second possibility for seborrhœa is that it may, and often does, become converted, so to speak, into eczema, that is to say, in place of the mere profuse desquamation of the scalp and altered secretion, you begin to get redness, irritation, heat and slight exudation, so that there are patches which, if not moist, easily become so when slightly scratched. When that is the case it generally extends and produces eczema of other parts, first of all behind the ears, very often in the nape of the neck, or on contiguous parts of the face, and then on those parts of the trunk where simple seborrhœa, as I have just said, occurs. After this it very often appears as an extension of

eczema, over a considerable part of the body and limbs, generally first on the arms, then on the legs. This is the affection which has been called by Unna, who first traced its connection with seborrhœa, *Eczema seborrhœicum*, or seborrhœic eczema; and, I think, the connecting of these two affections, and bringing them under one head, is certainly an important practical discovery. The only doubt I feel is whether he is right in calling the whole affection, from the beginning, eczema. It seems to me that the stage of seborrhœa and the stage of eczema can generally be distinguished.

I must say, however, that I do not, at present, see any means of always recognizing seborrhœic eczema on other parts of the body by its external characters. It appears to me that it cannot be distinguished generally from other forms of eczema, but is known, when the history can be traced, by following the course above described.

There is only one form of seborrhœic eczema, that which resembles psoriasis, which has certain definite characters, and though usually called psoriasis, may be distinguished. But as I have no case to show you, I will not pursue the subject further.

We might, perhaps, suggest—and I am rather in favour of this view myself—that, instead of calling all these affections, necessarily, seborrhœic eczema, it might be enough to call them cases of “descending eczema,” that is to say, that the head is the first part to be affected, and that the eczema slowly descends and comes out on other parts of the body; analogically we might also speak of “ascending eczema,” which is not very uncommon, that is a chronic eczema of the lower leg, which sometimes spreads to the rest of the body. There are reasons why I think the head at the one extremity and the legs at the other are likely to be centres for the extension in this way.

Another frequent consequence or sequela of scurfy head, which is not at all generally recognized, is the production of suppurating pimples or pustules about the face, neck, and neighbouring parts, in fact, the production of a form of acne. In some cases these pustules so produced are quite distinct from the typical kinds of acne, and if regarded as acne they ought to be called by the old name of acne pustulosa. In other cases this is not exactly the process. The order of things is that, when an acne vulgaris already exists, the result of the scurf on the head is to convert it into a suppurative acne. The seborrhœa on the

scalp is not the cause of the acne, but the cause of its suppuration. I believe this may be seen in the two kinds of acne, acne vulgaris and acne rosacea, for though they are absolutely different in origin, yet in the later stages of both the production of pustules is likely to occur. In addition, the boils in the nape of the neck, which are very common in people with this affection, arise, I believe, from something derived from the head. Other affections which, I think, may, in some cases, be assigned to the same cause, are "styes" of the eyelashes in children, and sometimes a mild form of conjunctivitis. In all such cases the scalp should be examined.

If asked how seborrhœa produces these changes, I think the explanation is something like this:—The mere scale production in the head is not, in itself, the cause of the secondary affections. The desquamation seems to be due to an alteration in the quality of the sebum. That is to say, if the sebum be normal, but in excessive quantity it forms an oleose seborrhœa; but if, on the other hand, it is not sufficiently fluid, it accumulates and forms thin, dry-looking scales. But that is not enough to cause a suppuration in any other part of the body. However, in these masses of scales there are enormous numbers of micrococci; and the explanation (which is not altogether theoretical) is that the scaly or scurfy head is a kind of nursery of bacteria, and here swarms of micrococci are cultivated and extend over the body, especially on the face and neck, where they set up suppuration determined by some slight local cause. It is very extraordinary, supposing suppuration to be due to the staphylococci which are so constantly found in pustules, that it should recur again and again on the face, which is being continually washed. It is difficult to believe in the establishment of a colony of bacteria on the surface of the face, but it is quite easy to believe that there may be colonies of bacteria on the scalp which get continually shaken down in the form of scurf to the face and neck, and cause points of suppuration. I have seen a good many cases in which suppurative or pustular acne is connected with this cause. One, the other day, was that of a little girl æt. 9. She had an exceedingly scurfy head. It had been a great trouble for some years. She was beginning to get on the nose, chin, and other parts of the face a regular pustular acne. At her age it is practically impossible a true developmental acne could have come on. Therefore I

think it was a local result of colonies of micrococci descending from the scalp.

Please note that in all this I say nothing about the cause of seborrhœa itself, whether primary or secondary, on which I express no opinion. Dr. Unna believes that he has discovered the cause of seborrhœic eczema in a particular species of micrococcus, with respect to which we await further particulars.

The foot also, for various reasons, is a great nursery of bacteria, even in people who give some attention to their feet. If neglected for a few days sufficient evidence is given that bacteria are flourishing there; and from the foot and lower leg also, I think they may spread over the body and set up irritation, producing secondary eruptions, which I have called "ascending eczema."

(To be concluded).

INTESTINAL OBSTRUCTION DUE TO GALL-STONES.

Paper read at East Kent Branch of the British Medical Association, Nov. 23, 1893, by

ARTHUR WELSFORD, M.D., F.R.O.S.,
Of Dover.

ALTHOUGH intestinal obstruction due to impacted gall-stones is not by any means a very rare condition, it is in the majority of cases most difficult to make a certain diagnosis. In acute cases the mortality is very high—a fatal result occurring in about 50 per cent. of all cases. Up to the end of 1892, at least 127 cases of fatal impaction have been recorded, and this number of cases allows us to make some generalizations about this particular form of obstruction which may prove of value in the consideration of any particular case.

Taking the average of these cases we find that death occurs on or about the tenth day, and if by that date the obstruction has not been relieved but little hope can be entertained of the ultimate recovery of the patient. The proportion of women to men among these cases is in the ratio of three to one, and in four-fifths of the cases death occurred between forty and sixty years of age.

Although impaction is by no means rare in the duodenum and jejunum, yet it usually occurs at the ileo-cæcal valve, and in the lower portion of

the small gut. Obstruction may be due to the large size of the calculus, or to a matting together of many small stones. A stone of size sufficient to obstruct the intestine must have reached the bowel through an internal fistula, and as the formation of such fistula is not, as a rule, attended with much suffering, it is therefore obvious why a history of biliary colic should be so frequently wanting in these cases.

In the diagnosis of the case stress must be laid upon a history of biliary colic or jaundice, or the passage of gall-stones at some time or other. The age and sex of the patient must also be taken into account; but when everything has been considered, the diagnosis in many cases must be largely conjectural.

In cases of gall-stone obstruction, the pain is nearly always excessive and continuous, with severe exacerbations; the vomiting is obstinate and distressing, and the paroxysms sudden and violent. The pain is also very apt to shift from place to place, and the shifting of the pain has been considered a characteristic feature of this form of obstruction. As a rule the course of the trouble is acute, but the development of the obstruction is not so sudden, nor is the pain and collapse so intense as is the case in internal strangulation. Unless peritonitis is present there is no marked tenderness, and meteorism is, as a rule, slight and confined to the upper part of the abdomen, if the obstruction is in the duodenum or jejunum. In these cases the vomiting always is very severe and soon becomes *feculent*, intolerance of liquids comes on rapidly, and urine is secreted but scantily. In other cases the duration of the obstruction may be more prolonged, and the symptoms less urgent.

Although there is great doubt whether a stone small enough to pass the common duct can ever give rise to obstruction, except in those cases in which obstruction is caused by a matting together of many small stones, or by a lodgment above a stricture or other narrowing of the intestine, yet even the presence of a small stone in the intestine is not always unattended with danger. In a post-mortem upon a lad of 18 who died with symptoms of perforative peritonitis, I found two small cholesterol calculi lodged in the vermiform appendix, which was ulcerated through at the tip. In another case a calculus which had safely passed through the length of the intestine, lodged just above the anus and gave rise to tenesmus and mucous diarrhoea

with constipation. The purgative medicines given her at the hospital the patient attended made matters worse, but an examination per anum soon revealed the cause of the trouble, and after the removal of the calculus the patient rapidly recovered.

In the vast majority of cases, however, small calculi pass through the intestine without giving rise to trouble.

After a stone has caused obstruction of the bowel, the condition of the patient is one of extreme gravity. In fortunate cases the stone may become dislodged, and passing down the intestine may be passed per anum without further difficulty; but this fortunate result does not occur in more than 50 per cent. of the cases of genuine obstruction. Death only too frequently is the result of this accident, and may be due to exhaustion from the pain and vomiting or from inanition, owing to the great difficulty experienced in feeding the patient.

The presence of the foreign body excites inflammation in the wall of the intestine, and leads gradually to perforation of the gut. Peritoneal adhesions may have time to form, and the escape of the bowel contents into the peritoneal cavity may be thus prevented. If the bowel becomes adherent to the abdominal wall, an abscess may form which may burst externally, and thus allow the stone to escape, leaving the patient with an artificial anus which may or may not spontaneously close.

If owing to the absence of formation of peritoneal adhesions, the bowel contents pass into the peritoneal cavity, suppurative peritonitis will ensue which will cause the death of the patient. This is, indeed, a common termination to the case.

Peritonitis may, however, be quite localized, and although the calculus may have passed into the peritoneal cavity, it may be so surrounded by adhesions that no escape of the bowel contents will take place. This condition occurred in the case of a lady of my acquaintance, who had her first attack of colic at 65. After six weeks of pyrexia with hepatic colic and jaundice, a small stone was passed, but soon afterwards a mass as large as a cocoanut was felt in the abdomen.

A diagnosis of cancer was made, but her medical attendant who had from the first diagnosed the case as one of perforation of the gut and ensuing localized peritonitis, was proved to have been right by the nearly complete recovery of the patient.

Unfortunately, diabetes, a not very infrequent sequela of gall-stones has since supervened.

Dr. Harley says that in some cases the calculus may become encysted in the intestine, and cause no further trouble.

Even after the calculus has been passed, and the obstruction relieved, the patient may suffer for some time from pyrexia, dry tongue, and profuse diarrhoea, which are due to an intestinal septicæmia. In one case the diarrhoea caused the patient's death fifteen days after the passage of the foreign body.

Cicatricial stricture of the ileum arising from ulceration set up by impacted and long retained gall-stones may give rise to serious trouble long after the stone has been passed.

Mr. Jonathan Hutchinson strongly deprecates all operative measures in these cases. He advises that the use of an anæsthetic should precede treatment. By the anæsthetic intestinal spasm will be relaxed, and the passage of the foreign body facilitated. When the patient is fully under it, the abdomen must be carefully palpated, and abdominal taxis employed for some time. Belladonna may afterwards be given in large doses, and if much pain be present the anæsthetic may be repeated and continued for some time. He says, "Very systematic attempts should be made under anæsthetics to get enemata to pass the ileo-cæcal valve. This clearly is the most rational method by which the slipping of the calculus may be facilitated. If the distal part of the gut be distended with fluid the stone would be almost certain to move onwards when such fluid escaped. Under no circumstances should an operation be done; for, however urgent the condition may seem, the probability is great that under these measures recovery will ensue."

Other surgeons, however, in severe cases, perform laparotomy. When the abdomen has been opened the gall-stone may be discovered lying in the gut, and an attempt may be made to manipulate it through the ileo-cæcal valve, after which it may be safely left to nature. If this fails, the bowel may be opened and the calculus removed. The intestine may then be stitched up and returned, or an artificial anus may be for a while established. Probably the condition of the gut will render the latter alternative imperative. In all cases of intestinal obstruction Opium is a great stand-by. Opium should never, in these cases, be given in the form of pills, but always in a liquid medium or

hypodermically. By it pain will be relieved, and intestinal spasm relaxed. Purgative medicines must be given very cautiously. A mild aperient may be administered after the patient is well under the influence of Opium. Enemata should be largely used instead, and they may be given with the least discomfort to the patient if a piece of soft rubber tubing, about six inches long, be attached to the nozzle of a Higginson's syringe, and after being well oiled be inserted some little way into the rectum. The diet in all these cases must be very carefully attended to.

The patient's strength must be supported by nutritive enemata, and in acute cases nothing whatever should be allowed by the mouth except a little ice to suck, to allay the sickness. Death from exhaustion is, in the majority of cases, due to inanition. If the vomiting is not very severe, some highly concentrated peptonized liquid food may also be allowed. Thirst must be combated by the use of rectal injections. In chronic cases more liquid food may be allowed, and Opium and enemata must be freely resorted to. Mr. Treves says in these cases lumbar colotomy must be regarded more as a surgical misfortune than as a recognized method of treatment.

To conclude this account of gall-stone obstruction, I should like to draw attention to a very rare condition of obstruction simulating by its symptoms cancer of the pylorus. Owing to the situation of the gall-stone the duodenum has become compressed, and a dilatation of the stomach has resulted. In these cases a hard pyloric mass may be felt, and the symptoms of pyloric obstruction are so well marked that it is difficult to see how the condition can be distinguished from cancer before death. I believe only eight or nine such cases have been recorded. They will be found scattered in various journals. Among these are the following:—In 1889 Schreiber recorded the case of a woman 29 years of age, who, for six months, suffered with symptoms of pyloric obstruction. This was thought to be due to the contraction of a simple ulcer, and operative relief was advised. During the operation a large gall-stone was found in the stomach which had blocked the pyloric opening. After the removal of the gall-stone a complete cure was effected, which has lasted to the present time.

A case was recorded by Dr. Hale White, in 1885, of a woman who died with symptoms of pyloric obstruction, which were attributed to malignant disease.

P. M. The gall-bladder, which was full of gall-stones, was found to be adherent to the pylorus, which was so much thickened that obstruction had ensued. The cystic, common, and hepatic ducts were normal. There was a small sac, as large as a good-sized pea, which was situated in the thickness of the pylorus, and contained several minute gall-stones. From this a sinus opened by a minute opening into the stomach at its junction with the duodenum. The thickened gall-bladder was adherent to the pylorus opposite this sac.

What appears to have happened in this case is as follows:—Some gall-stones ulcerated through from the gall-bladder into the thickness of the pylorus, where they formed a sac for themselves. The communication between the gall-bladder and the sac subsequently becoming closed, the sac discharged itself into the stomach. The irritation of the foreign bodies set up a hyperplasia of the fibrous and muscular tissues of the pylorus. All the other organs were healthy.

Naunya relates the case of a man, æt. 57, who died with the most typical symptoms of pyloric obstruction, which had lasted about two years. The stomach reached the symphysis pubis, and a pyloric tumour was very evident. At the post-mortem no cancer was found, but the gall-bladder, which was filled with a large stone, had become adherent to the pylorus, and had reduced its lumen to a minute canal.

In conclusion, I may relate the following remarkable case, recorded by Miles. A woman, æt. 56, suffered for two years with symptoms of pyloric obstruction. The complexion was sallow, and the woman was emaciated. Vomiting of a brownish acid matter, which the microscope showed consisted of partially digested food with an abundance of sarcinæ and torulæ, was very frequent, and abundant to the amount of three or four pints daily. Suddenly she was seized with violent palpitation and intense pain over the stomach and right side, with collapse. This was followed by violent sickness, and she vomited a gall-stone seven-tenths of an inch by five-tenths of an inch. A fortnight later another gall-stone was vomited. After this she completely recovered, and the vomiting and other symptoms disappeared.

For Dry Bronchitis, to promote Secretion.

R Apomorphine Hydrochlor.... gr. j
Syr. Simplex 3ij
Aq. ad 3xij
M. Ft. mist. Sig.: 5j every two hours.

THERAPEUTICAL NOTES AND FORMULÆ.

Delirium Tremens.—Dr. Manchot strongly recommends Chloralamide in preference to Chloral. In doses of gr. xlv to 3ij. It acts very well, and without the unpleasant after effects of Chloral.

Munch. Med. Wochensch., 17, 1893.

Dr. F. Powers has tried with success the application of Collodion to the prepuce in cases of enuresis in boys. Before the child goes to bed a layer is applied in such a way as to close the preputial orifice; should the child wish to micturate the seal can be easily removed with the finger. Dr. Powers claims to have cured several cases within a fortnight by this simple plan.

Wiener Med. Presse, 17, 1893.

Cystitis.—Okev-Blom, in acute and in chronic cystitis, praises the injection of Iodoform-Æther-Oil into the organ every second or third day. The mixture is in the proportion of Iodoform 1 part, Æther and Olive Oil of each 7 parts; about 3iiss to 3ij to be used for each injection.

Annal. des mal. des org. genito urin.

Dr. Meynier recommends hourly doses of Salicylate of Soda in cases of acute gonorrhœal inflammation about the neck of the bladder. The drug is given in 20 grain doses for six doses at least.

New York Med. Record.

For Hyperidrosis (excessive sweating of the feet):—

R Naphthol 3j
Glycerine 3ij
Alcohol to 3iv

M. Signa: To bathe the feet morning and evening.

Afterwards dust the feet with the following:—

R Naphthol. Pulv. gr. xxx
Amyl. Pulv. 3vj

M. Ft. pulv. Signa: "Dusting powder."

The following powder may be dusted into the boots every morning:—

R Pot. Permang.... .. gr. xlv
Sodæ Salicyl. gr. xxx
Bismuthi Subnit. ... 3iiss
Talc. Pulv. 3j

M. Ft. pulv.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 20, 1893.

A CLINICAL LECTURE

ON

ACUTE ASCENDING MYELITIS.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, Bloomsbury,

By W. R. GOWERS, M.D., F.R.S.

GENTLEMEN,—Before we proceed to the special subject which we are to consider to-day, I propose to describe to you a case I saw late last evening. There are many diseases which, because they are rare, or for other reasons, seldom come under the student's notice, and although description can never take the place of personal observation, it may be useful if the latter is impossible, and it may be the more useful if a case has been recently seen, and its facts are fresh and vivid in the narrator's mind.

The patient whom I saw (with Dr. Parnell and Dr. Grayling, of Forest Hill) was a lad of 19, paralysed almost completely from head to foot, pale, with the distressing struggle for breath that comes when breathing power is getting less and less. The story of the case is as follows :

A month ago he had his first lapse from virtue, and the consequence was—not a rare one—an attack of gonorrhœa ; it was treated, and he recovered in a fortnight. He had not previously been in good health, having been over-worked at night. A little more than three weeks after the onset of the gonorrhœa—about a week after its termination, and six days before I saw him—his legs, one day, became weak, he stumbled on the stairs, and in a few hours the weakness became considerable. The next day his abdominal muscles were feeble, and the legs powerless, but with excessive knee-jerks (which disappeared two days later). On the following day his arms began to lose strength. This weakness increased and extended during the next three days, and he became febrile. When I saw him last night his state was this : He was sitting, half upright, in a chair, breathing, as I have said, with difficulty, by means of his diaphragm and lower intercostal muscles, at the rate of 56 respirations per minute, the deficiency in quantity of air inhaled having to be made up by increased fre-

quency of respiration. His temperature was raised to 103°. His legs were absolutely powerless, without a trace of knee-jerk—flaccid palsy. From the right sole there was no reflex, but a touch on the left caused a considerable movement of the foot ; there was no abdominal reflex, but a stroke on the skin left the bright red line, lasting a long time, which shows acute disturbance of vascular innervation. He could just flex his fingers, and that was all the movement left in his arms. The abdominal and trunk muscles were paralysed, except the lower third of the intercostals and the diaphragm. The upper part of his thorax was motionless, and the sterno-mastoids were also powerless. He could swallow, and he could just speak, although with difficulty. The left side of his face moved less than the right ; the left masseter contracted a little less than the right, and movement of the eyes was accompanied with brief but wide nystagmus, greater on movement to the left side than to the right. Without any discoverable cause—without any hot application or the like—there had developed, within a few hours, a very large bulla over the inner side of the right ankle.

It was thus a case of acute ascending paralysis, using the words in a symptomatic sense, and it had naturally been regarded as a case of that mysterious disease to which the term is specially appropriated, and which is also called "Landry's paralysis." I think that this reasonable conclusion is very near the truth ; but there was evidence of more than we have in that disease. Indeed, the case is particularly instructive from the point of diagnosis. "Acute ascending paralysis" depends apparently on a peculiar arrest of central, and perhaps, peripheral, function, by a peculiar blood state. In it there is no pyrexia ; the loss of function presents a perfect bilateral symmetry, never transgressed by such a deviation as the preservation of the plantar reflex on one side when it is lost on the other. The course of the symptoms is uniform, the knee-jerk is lost from the first, and never excessive at the beginning, as in this case. Although it is said that some parts may be passed over in the upward march of the palsy, as, in this patient, were the lower intercostal muscles, yet I think it is doubtful whether this is really true of Landry's paralysis, and whether the assertion has not been due to

cases—such as this—which may so readily be mistaken for that affection. The symptoms pointed to a derangement of the functions of the spinal cord, slightly but distinctly irregular, both in time and place. There is no doubt that Landry's paralysis, as I have just said, is the effect of a toxæmic state on the nerve functions; but all toxæmic conditions, in their isolated action, cause *symmetrical* arrest of function; and the difference we had here in different parts and at different times, showed that there was an irregularity in the cause of the symptoms,—something more irregular than the simple inhibition of a blood state. The early excess of the knee-jerk, together with the difference between the two plantar reflexes, and the nystagmus, pointed to a central affection; the first of these suggested a process in the spinal cord beginning, and at first most intense, above the lumbar enlargement; while the pyrexia, developing late in the course of the affection, was evidence of inflammation rather than of a primary blood state. Had the fever been due simply to the cause of the symptom, it would have occurred early. Its indication—that there was inflammation—agreed with the spinal symptoms. However set up, acute inflammation in the nerve centres always tends to more or less irregular and random influence. This is no doubt largely determined by the participation of the vessels in the process, and by other causes, such as a slightly greater intensity in one tract of grey matter in which it may spread, or the local influence of a small inflammatory extravasation. Hence inflammation, when set up, extends in a more or less random manner, and its symptoms are often irregular, at least in some degree.

The significance of these indications is that, in this patient, we had to deal with acute ascending myelitis. This is the condition between which and the special "acute ascending paralysis" there is generally most difficulty in diagnosis. Although the initial excess of the knee-jerk suggests that the inflammation began above the lumbar enlargement, and that the centres in the latter were merely irritated at that stage, yet the complete palsy of the legs shows, however, that even then the damage to the cord above the lumbar enlargement must have been considerable. As the inflammation spread, upwards and downwards, the damage to the centres in the grey matter of the lumbar enlargement caused absolute loss of the knee-jerk, but it would seem that, even to the

last, the lowest part of the cord on the left side was not completely involved, so that a plantar reflex could still be obtained. Trophic disturbance, such as the large bulla on the side of the heel, does not occur in Landry's paralysis: it is an indication of an intensely irritative state of the nerves, transmitted to them from the spinal cord through the posterior roots. Although the posterior nerve roots conduct impressions upwards they transmit nutritional influences downwards. This transmission is, indeed, probably by their own nutrition; the disorder in their molecules passes to the tissues in which they end. This bulla, in connection with the other symptom, showed that there was an intense irritative inflammation in the spinal cord. It was on the side on which the plantar reflex was abolished—that is, on the side on which the inflammation of the grey matter, in the lowest part of the cord, was most intense. The lad's condition did not allow of our ascertaining exactly the state of sensation.

But, in the lessened movement of the face, of the masseter, and of the eyeballs, trifling as all were, there was evidence that the inflammation was spreading upwards into the pons. It seemed to have passed the medulla, except that the heart-sounds were unnaturally short, with a peculiar character, seldom met with except in acute disturbance of innervation, as if the cardiac centre were becoming affected.

I have said that the opinion that he was suffering from Landry's paralysis was probably not far from the truth. There is no doubt that Landry's paralysis is due to an acute blood poison acting on the nerve centres, from below upward. All present knowledge leads us to ascribe an acute inflammation of similar course to a like cause. Both maladies occur under analogous conditions. It is probably only a question of a slight degree of difference in the nature of the blood poison, whether it simply arrests function and causes slight nutritional changes, or whether it sets up actual inflammation. Measles may be followed by typical Landry's paralysis, without evidence of inflammation; and measles may also be followed by an intense spreading myelitis. We are learning to see more and more distinctly the profound causal influence of toxic blood states in producing inflammation of the spinal cord as well as of the peripheral nerves, and it is most important to recognize the fact that allied blood states seem to cause one, or the other, or both.

At present, multiple neuritis has the hold on professional thought which novelty always entails, and central diseases are sometimes overlooked. I have lately read some descriptions of acute fatal "multiple neuritis" (without autopsy), due to toxæmia, which I am sure were cases of ascending myelitis.

Among these blood states which act on the nervous system, some of the most important are those connected with acute specific diseases—the diseases that are due to an organismal virus. The question was put to me in this case, Was the lad's malady connected with the gonorrhœa? I do not know that anything of the kind has been hitherto observed as a result of gonorrhœa, but a causal relation between the two is highly probable. We know how such states are produced by various acute specific diseases; and we know that the poison, the organized poison, of gonorrhœa may induce a subsequent toxæmic state which is manifested by the arthritis of "gonorrhœal rheumatism." Let us consider, for a moment, what this means.

The organisms of such diseases seem to cause these consequences, in most instances, by producing a chemical organic poison in the blood, and analogy makes it highly probable that such an agent is the cause of the arthritis. But we know that such products can act on the nervous system, peripheral and central, inducing grave disturbance of function, and definite, often severe, inflammation; it is therefore not surprising to find that, in some individuals, especially predisposed, the gonorrhœal virus should cause an after-poison capable of producing the terrible effect which was seen in this case. Let me remind you of a fact, which I may have mentioned before and may have to mention again, because it is one of the cardinal facts of medical knowledge, throwing light on many problems, and, among others, on the influence of predispositions. It is the fact ascertained by the admirable researches of Dr. Sidney Martin on the process by which diphtheritic paralysis is produced. He seems to have conclusively proved this,—that this paralysis is due to a poison of organic, chemical nature; that the poison is not produced directly by the diphtheritic organisms, but that these generate a particular chemical substance of the nature of a ferment, which acts upon albuminous materials ("albuminoses") in the body, especially in the spleen, and converts them into a poison which has

this intense action on certain nerve structures. It is probable that the mechanism is similar by which many other specific organisms give rise to toxic agents. You can readily understand that if a person, has previously been in bad health, as this lad had been, there should be some slight defect in the chemical constitution of such substances as the albuminoses of the spleen, etc., and a very slight defect may render the product of such a ferment different from that which would result in perfect previous health. A very slight difference in constitution, a little more or less of one element, may change a toxic substance into a harmless one, and *vice versa*. Thus we can understand how it is that the same causal influence shall have but little effect in one individual, and shall have a profound effect in another, especially in another who has been in conspicuously bad health. We can understand, for instance, that the organisms of gonorrhœa shall, in one person, give rise to a toxic chemical material which acts on the joints, and causes the arthritis of "gonorrhœal rheumatism," while, in another, the poison produced shall act on the spinal cord and set up acute myelitis as in this case.

The lad was in a state in which death seemed inevitable. In "acute ascending paralysis" (Landry's paralysis), as I have said, we have only an arrest of function by the blood state; in inflammation we have also a process of organic change, in the vessels and the tissues, which tends to progress even apart from its cause. The mere arrest of function, once stopped, may not increase, and may be followed by steady recovery; but the effects of inflammatory damage must persist, and the process itself may continue for a time after the blood state which has caused it ceases to act. Hence, I fear that the chances for the poor fellow's life are very small. I doubt, indeed, whether he is alive now.

Although the expectation of saving life may be absent, and even the hope of doing so can find no room in reason, we are always under the compulsion of striving to achieve that which we may not be able to think possible. Our knowledge is never certain—even our possibilities may be incorrect, and so our hopelessness may be wrong. We must act in spite of our anticipations. Therefore, it will be right for you to ask, what did I suggest should be done for him?

As far as I have been able to see, there are only two agents which have any considerable influence

upon toxic blood states of this kind. One is mercury. I believe the influence of mercury in syphilis is only one instance of its power over organisms. It is the most striking and perhaps the only one surely known to us, but it is probably not the only one, and is not likely to be the only one. Some years ago, a discovery was made in some researches at the Brown Institution which has been almost unnoticed since,—that the fatal effects of a certain inoculation (I think with the poison of anthrax) could be prevented by mercury. Its influence in other diseases of like nature is supported by many facts. That on inflammation, which few can doubt, may be connected with its effect on blood states. So I advised that mercury should be rubbed in—a drachm of mercurial ointment every four hours. In such a case, however, you must “hit right and left”; there is not an hour to lose; you must do anything and everything you can, always provided you do no harm. In septicæmia, such septicæmia as occurs after childbirth, for instance, the agent which has seemed to me effective, and which, as far as I could judge, has certainly saved life, is perchloride of iron in full doses. So I advised that every three hours 20 minims of the tincture of perchloride of iron should be given. The only other opportunity for treatment, beyond ordinary stimulation, was this. He might die from failure of the functions of the medulla; they might fail from the influence of disturbance of other related parts, or from an influence which was not an actual inflammation; it was just possible that if they could be kept active he might tide over a period of danger. The measure that has seemed to me to have most influence in stimulating and keeping up the functions of the medulla, is the hypodermic injection of a small quantity of strychnia, one-eightieth of a grain, together with a very minute, stimulant, dose of morphia, about one-thirty-sixth of a grain, repeated every two hours if necessary. Of course, if the failure of function were due to invasion by inflammation, this could have no effect.

But there was no reasonable ground for hope that the treatment would be effective. The cause is more likely to be a chemical substance than organisms, and we have even less power over the former than we have over the living agents of disease. Had the symptoms been due merely to the restraining influence on function exerted by the blood state, we could have felt a slender hope that its maximum might have been reached. But inflamma-

tion, to whatever due, is a process with its own effects—proportioned to its initial energy, but locally independent of its origin—certain, at such a stage, to cause more destruction. Already at the limit of the functions essential for life, it could scarcely stay there; the passage of that limit seemed inevitable, and with it the thread of life would break. Next week I shall doubtless be able to tell you.

[In his lecture the following week Dr. Gowers mentioned the sequel. The boy lived only six hours. A post-mortem examination, made by Dr. Arkle, revealed characteristic signs of myelitis, most intense at the place at which the inflammation had been supposed to begin—the lower part of the dorsal region, just above the lumbar enlargement. There, indeed, it had caused the front of the cord to be bulged forwards in an unusual manner by the swelling and softening of its substance. There was also some softening in the mid-dorsal and lower cervical region, where the grey substance was hæmorrhagic, almost diffuent. Culture-investigation for organisms has been commenced, and the pathological results of these and of the microscopical examination of the cord, will be published by Dr. Arkle.]

A CLINICAL LECTURE

ON

HÆMORRHOIDS.

Delivered at the London Hospital, Nov. 8th, 1893, by

FREDERICK TREVES, F.R.C.S.,

Surgeon to the Hospital.

I do not propose, Gentlemen, to do more than draw attention to some of the leading features of this very common trouble—hæmorrhoids. There is a case now in the hospital which will be operated on on Friday, when I propose to carry out what is known as Whitehead's operation, so that you can see at least one method of dealing with this trouble.

In considering the points which, I think, may be, perhaps, most practical, we will begin by asking, What are the causes of hæmorrhoids?

Well, the first and chief cause is an anatomical

one, and that involves a very common error. You ask a student, What is the blood supply of the rectum? He tells you, The superior hæmorrhoidal artery from the inferior mesenteric, the middle hæmorrhoidal from the internal iliac, and the inferior hæmorrhoidal from the internal pudic; and it is almost inevitable to find it assumed that it is the inferior hæmorrhoidal, as being the vessel nearest the anus, which is most concerned with this trouble. Remember that it is the superior hæmorrhoidal vessel, and the superior hæmorrhoidal vessel alone, that has to do primarily with hæmorrhoids. Speaking of "vessel" in this connection, I mean the vein. Some of the details of that vessel you must appreciate.

Let me remind you how the lower part of the rectum is developed. The greater part of the rectum is formed by a protrusion coming down from the hind gut in the abdomen. That protrusion as it comes towards the anus, or where the anus will be, carries its own blood-vessel (the superior hæmorrhoidal) with it. A time comes when a little involution of the integument takes place. That involution brings its vessels with it, the middle and inferior hæmorrhoidal arteries. The two structures meet, and so we have the actual rectum as a tube. The superior hæmorrhoidal artery supplies the mucous membrane of the rectum down to the very integument; an injection of that artery will inject the whole of the mucous membrane of the rectum to the skin.

Remember, too, there are no valves in the superior hæmorrhoidal vein, inject that, and you will inject every square line of the mucous membrane of the rectum in its entirety.

What, then, do the middle and inferior hæmorrhoidal arteries do? They do little except supply the soft parts outside the true rectum, and they may be disregarded altogether, I mention them because of the common error with which they are associated. This disposition of the vessels has been pointed out by Ball.

It is often stated in the text-books that piles are common because they occur at a point where two circulations meet, the portal and the systemic. This has nothing whatever to with the causation. Ball proved that you cannot inject either the middle or inferior hæmorrhoidal vessel from the superior. Throw a fine injection into the superior hæmorrhoidal artery, and you can inject the whole rectum, but no injection will pass from

the superior into the inferior or middle vessel. The same applies to the veins.

Let us remember, then, that it is one set of vessels only that is concerned in the anatomy of hæmorrhoids.

We must be somewhat particular as to the method in which these vessels are disposed. You must be familiar with the arrangement of the veins in the lowest part of the rectum to understand piles. In the upper bowel generally the vessels are arranged in a circular manner at right angles to the long axis of the gut. But see what happens in the rectum. At the margin of the anus you find a number of dilated vessels, about twelve in number, arranged circularly at the very verge. You will find them figured in most text-books. They are connected by transverse branches, and can be seen very well in any special preparation. This circle of dilated vessels (mind you, normally dilated) ends in a number of straight veins. In no other part of the intestine is this condition to be found, all the veins of the intestine are arranged in exactly the opposite direction. These veins run up the bowel pretty well perfectly straight. As a matter of fact they run up in the columnæ recti, are in the sub-mucous tissue, and are generally about six in number. Having gone a certain distance they do a curious thing. They all go through holes in the muscular coat, and then, of course, cease to be sub-mucous. This sudden diversion takes place some three inches from the anus. It is necessary to understand this arrangement to appreciate Whitehead's operation. You may say what is the use of remembering all this. Well, when you do Whitehead's operation, you have to cut all these vertical vessels; and, therefore, it is important to keep in mind their number and arrangement. What you say of the veins you may say of the arteries. The arteries are about six in number. They come down outside the bowel until they are three inches from the anus, they then pass through holes in the muscular coat and become entirely submucous, and they also end in transverse inosculating branches at the margin of the anus.

The veins have no valves, and little support of any kind.

These, then, are definite anatomical reasons for piles which you may give, and they come under these seven headings:

- (1) The blood is returned against gravity.
- (2) The veins have no valves.

(3) The veins pass through muscular tissue.

(A somewhat unfortunate thing.)

(4) They are affected by every form of portal obstruction.

(5) They are influenced by the erect posture, either sitting or standing. I need not say that a disease similar to hæmorrhoids is found in no other animal than man. It has been very well urged that the erect posture has more to do with hæmorrhoids than has any other factor.

(6) There is an extraordinary lack of support to the veins.

(7) The mechanical effect of defæcation has to be regarded. That would take too long to follow out; but you can quite understand how it would act. In the passing of a motion with a certain amount of straining what is the result? Look how the veins of the portal system must be engorged by the contraction of the abdominal muscles, and what effect upon these veins would you expect that a hard fæcal mass would have as it passes down through the lower part of the rectum; and is it not a fact that piles are first noticed in connection with the act of defæcation?

The other causes I will slur over, because that is one of the things you are not likely to be indefinite about;—the question of age (they are much more common in middle life); the question of heredity (which cannot be disputed); the effects of pressure (as shown by tumours, by the gravid uterus, by any form of portal congestion); and then the noteworthy effect of constipation and the use of aperients.

Do not trouble about the complex methods of classifying piles, because they have little clinical value; and are of small moment as regards treatment.

Piles can be most simply grouped in this way. There is

- (1) The external pile.
- (2) The internal pile.
- (3) The marginal pile.

Of course we all know what we are speaking about when we talk of "a pile." It is a varicose vein; that is all.

The external pile is covered with skin.

The internal pile is covered with mucous membrane.

The marginal pile is covered with both.

Do not get led into talking of piles within and without the sphincter. That is mere folly. If the pile is entirely within or beyond the internal

sphincter, goodness knows where it will be; it will be something more than an internal pile. The term "external" pile has only reference to its being external to the anal margin; the "internal" pile is covered by mucous membrane; and is submucous: and these differences come out in treatment. The marginal pile cannot be said to be either inside or outside the anus; it is on the margin itself, covered both by skin and mucous membrane. It is therefore a pile occupying both a subcutaneous and a submucous situation.

With regard to the external pile it is very simple.

Now, does a man come to a doctor complaining of external piles? Very seldom, if they are simple. He comes, as a rule, when a certain accident has happened, viz., when the piles have become thrombosed.

Just consider then the external pile

- (a) before thrombosis;
- (b) during thrombosis;
- (c) after thrombosis.

The practical value of that you will see in a moment.

(a) Try to appreciate the condition of an external pile that has done nothing but exist. It is a varicose vein, outside the margin of the anus, covered by skin. What trouble does it give? Well, it is hardly to be put into words. The patient, perhaps, has a little sense of pain on defæcation, a slight sense of fulness at the margin of the anus when he defæcates, and a lack of relief after the act of defæcation. Some patients say they feel disposed to lie down after the bowels have acted, or to apply pressure to the part; and on cross-examination they have little more to say about it. So the pile before thrombosis gives practically little or no trouble.

The treatment of external pile before thrombosis is simply palliative. We shall come to this immediately.

(b) As for the pile *during thrombosis*, what has taken place? The pile is a varicose vein, and does just what any other varicose vein may do—it may become inflamed. As the result of the phlebitis it becomes thrombosed and filled with clot, and, curiously enough, becomes in a rough sort of way cured. But, unfortunately, that is not brought about without infinite distress. The patient has for a long time had a vague idea that he has piles. One day something happens. There has been constipation, or the passage of a hard motion, or an alcoholic bout, which has confused details a little.

Anyhow, something has taken place. There is enormous pain at the anus; the parts are indescribably tender, red, hot, swollen, œdematous. The man does not dare think of defæcating. He has pain in his back, some fever, malaise, and a coated tongue. If you had asked a doctor twenty-five years ago what was the matter with the patient, you would have been told he had "an attack of the piles." The attack of the piles in ancient days included many things, and among them the thrombosis of an external pile. There are other symptoms, notably spasm of the sphincter.

What are you to do in such a case? Rest, low diet, and cold are to be employed; in other words, the man stops in bed, keeps to a scanty and elementary diet, and has applied to the part either an icebag or an evaporating lotion.

What are you to do to the pile itself? Either incise it or excise it—it matters little which. If you incise it you make a cut into it, and taking your two thumb-nails turn out the blood clot. If you do not like to turn it out in this way you may simply cut the whole excrescence off, and it does not bleed because it is filled up by a thrombus. That is Nature's method of curing a pile; a method exactly imitated by the injection of Carbolic Acid, which produces thrombosis.

(c) What happens *after thrombosis*? A man has had his attack of piles; he has rested, or has not rested with it; he has had treatment, or he has not: what is left behind? There is left behind the covering of the pile, a pendulous lump of skin, within which is a closed, wasted, varicose vein, which was at one time blocked up with a clot; the clot has gone, and the vein, as a patent tube, is gone: there is only left a skin tag. Well, what is going to happen to it? It is a thing of comparatively low vitality, and it contains still some inflammable material; it is liable to become inflamed, ulcerated, and œdematous. In some text-books you find a special form of hæmorrhoid spoken of as the œdematous pile. That is simply one of these skin tags, or an external pile after thrombosis, which has got into trouble.

What are you to do with it after it is thrombosed, and is in the condition of a skin tag? There is only one thing to be done. Cut it off.

With regard to *internal piles*, they are covered by mucous membrane, and are therefore sub-mucous. When perfectly uncomplicated little can be made out of them by a digital examination of the rectum; they are likely to bleed, and are often

called "bleeding piles." If they are old the mucous membrane is likely to become thickened and skin-like; and those you can feel by the finger. Remember always they are simply varicose veins under the mucous membrane. Varices need not be limited to a great vein. A varicose condition may restrict itself to the great saphenous vein, but it does not always do so; it may spread to the venous capillaries about the knee and thigh. So in the rectum; if the smaller venules are involved you have a pile to which the special name of "nævroid pile" has been given.

The symptoms you know well enough. There is always pain, and much of it is reflex pain in the back, pain about the loins. There is always bleeding, and this bleeding is at first noticed only at stool, then at other times. There is the sense as of a foreign body in the bowel; the patient feels perfectly certain there is something more than piles, something growing in the rectum, a polypus or the like. There is also tenesmus. The patient says that if he takes an aperient he will feel disposed to go to the closet ten or a dozen times. There is spasm of the sphincter, which is very troublesome at night. The piles protrude on defæcation, and there is some mucous discharge. That is the common story with which a patient comes to you.

The external pile is liable practically to only one trouble, but the internal pile is liable to accidents, which are in practice often confused one with the other. These internal piles may become strangulated on the one hand, or on the other they may become inflamed or actually gangrenous. In the former case, the piles are forced out at the anus, the sphincter contracts on them, and they won't go back, they become strangulated exactly as a hernia does. Or, on the other hand, they do not so protrude, but they become inflamed, and thereby thrombosed, and so vigorous may the inflammation be, that (being placed under the most disadvantageous conditions as regards blood-supply) they slough and become gangrenous. Strangulation is bad enough, but gangrene of internal piles is worse.

Now, as to a few questions about *treatment*. I shall say no more about external piles, because we have done with them. We shall refer merely to internal piles, the more real trouble.

There is, first, the removal of the cause. It may be removed. There is little use treating a pregnant woman for hæmorrhoids. They will go when the

cause of the hæmorrhoids goes, and the uterus is emptied. The same applies to ovarian tumours and the like.

The next thing is exercise. A sedentary life is one specially favourable to the development of piles. You may not credit the statement, but this one element of treatment will often cure piles.

Then there is diet. This consists of the ordinary diet advised for those who are the subjects of constipation.

Many patients cannot jump back to a normal condition of the intestine at once. They must take an aperient. Aloes given in a small dose of the extract answers well. Many old subjects of hæmorrhoids will take nothing else. A grain and a half of Extract of Aloes, and a saline aperient in the morning in the form of some mineral water is a common formula. Another suitable aperient is Sulphur in the form of the Compound Liquorice Powder of the Prussian Pharmacopœia, and a third is Cascara. That would represent, I think, the order of merit of these three aperients. Then the bowels should, if possible, be opened at night or in the evening, so that the patient can rest through the period of sleep with the bowel empty and the veins in the best position to empty themselves. In any case it is well that the patient should lie down and rest after every act of defæcation.

If there be much anæmia from loss of blood, Iron is needed, and no form answers better than Flitwick water or Levico water.

With regard to applications what are you to do? We cannot go on for ever ordering an ointment of Opium and Galls. The chief interest of that preparation lies in its antiquity.

A preparation advised by Dr. Ball I think you will find perhaps the best application, combining the two things you most want—a sedative or narcotic, and an astringent. It is as follows:—

Morphia, 10 grains; Extract of Belladonna and Tannic Acid, a drachm of each; an ounce of Lanoline, and an ounce of Vaseline.

That, of course, is best suited for outwardly presenting piles; but those that are within the margin of the anus are better treated in another way.

If you use an injection, what is the injection to be? A good injection is that of Sulphate of Iron, 10 grains to the ounce. You inject one ounce with a glass syringe. This, given after the bowel has acted, will usually be retained, and is admirable; when there is bleeding it is perfect. The injection should be warm.

The next best injection, or one that is better sometimes, is Hamamelis, which is very simple to use: ten drops of the Extract of Hamamelis to one drachm of water. This can be used at any time without inconvenience to the patient. It is administered by a Glycerine enema syringe after each stool.

Another very good remedy is Hazeline, the active principle of Hamamelis. It sometimes answers better than Hamamelis. You use one drachm of Hazeline with half a drachm of warm water, administering it by means of a Glycerine enema syringe after each motion. Of course, it may be administered in the form of a suppository, which brings the fluid in contact with the bowel after the wax of the suppository has melted, or as an ointment; but the best plan is by injection.

With regard to operations. About many of them I do not think you need trouble. I shall mention last those that appear to be the more valuable; the others I shall merely enumerate.

(1) *Caustic*, in the form of Nitric Acid. When I was a student it was a common method of treating piles. I don't suppose it is much used in the present day. It is of some value in the case of small, florid, irritable piles. It has to be applied through a speculum; the parts have to be prepared with ointment; and, as you cannot well limit the action of the acid, it is a little dangerous.

(2) *The injection of fluids into the pile*, I suppose, you would be disposed to ridicule. There is a certain class of patient who will not submit to any kind of operation. Is there then any reasonable method of treating piles without an anæsthetic?—for it is really the anæsthetic which is usually the bar. How do piles cure themselves? By thrombosis often. And why not induce thrombosis? With Carbolic Acid this may be done with no little success. By this method piles can and may be cured without an anæsthetic or an operation. You use a solution advised by Mr. Swinford Edwards:

R. Carbolic Acid gr.xij
Glycerine ʒj
Water ʒj

By means of a hypodermic syringe three to four drops are injected into the centre of each pile. No after treatment is requisite. There is no confinement in bed. Mr. Swinford Edwards, who is an advocate of this treatment, will apply it to a patient in his consulting room and let him immediately go home. I do not say there is no pain, but it is often surprising how little is complained of; it is followed by an

amount of success you would hardly expect. The results, as detailed in Mr. Swinford Edwards' admirable book, are not to be disregarded.

(3) *Dilatation of the sphincter* with bougies is not of very much value. It has been assumed to act by the rest that is necessary both before and after the bougie is passed. A large bougie, No. 12, is used. How it acts it is difficult to say.

(4) *Electrolysis* is a bad method, being both uncertain and dangerous.

(5) *Crushing* is a method of treatment with little to recommend it. It is uncouth and rough, and can certainly be replaced by better methods.

We are now brought down to two methods. One is the treatment by

(6) *Ligature*. The other is

(7) *Whitehead's operation*.

So far as I know these represent the best methods of treating piles. Of course, you must use discretion in selecting your cases. If you have a woman who is neurotic, wasted, worn out with the pain and discomfort of piles, and very anæmic, you would not dream of performing Whitehead's operation. That may take from half-an-hour to one-and-a-quarter, and the amount of blood lost is always considerable. But the result is good. The patient gets up in a fortnight or ten days apparently well. There is no time to go into the details of the operation. Many little details have to be attended to. There is no incontinence; and I have never seen any case of stricture resulting from it.

A great thing in operation cases of piles is the after-treatment. The treatment consists in absolute rest in bed; very moderate diet; Opium; the bowels to be opened on the fourth day and every day afterwards; and some support to be given to the anus. To effect this support T bandages may be used; but the sanitary towels used by women are better, especially those consisting of some antiseptic preparation of wood wool. They are very comfortable, and are burnt after use. Why put on any bandage at all? Well, dispense with it and hear what the patient has to say. You will find that the patient *must* have a support. Put the patient back to bed without the Iodoform, without the suppository, and he may not complain; but put him back without any support, and he will lie for hours supporting the perinæum with his hand. A man will adjust his T bandage several times a day to secure this support. This is evidence which will upset much theory.

ON SEBORRHŒA AND ITS CONSEQUENCES.

A Lecture for the London Post-Graduate Course, delivered at the Hospital for Diseases of the Skin, Blackfriars, Nov. 7th, 1893,

By J. F. PAYNE, M.D., F.R.O.P.,

Physician to St. Thomas's Hospital, and to the Hospital for Diseases of the Skin.

(Concluded from p. 109.)

We must now speak of *treatment*. If seborrhœa produces these results, it is certainly a thing worth attending to. The treatment of seborrhœa has been a good deal written about, and we frequently see letters in the medical journals asking how to treat dandruff of the scalp.

The first way in which people generally try to treat it is by washing their heads often, but it is generally found that this does not succeed, because though it momentarily relieves the scalp from the abundance of scales, they quickly return, and even more abundantly. The next remedy generally tried is some alkaline application. The most popular remedy of this kind is perhaps Borax. Borax acts in the same way as other alkalies, such as Carbonate of Soda, etc. They clear off the scurf with great rapidity for a short time, but it always returns. Lime water mixed with oil, the "lime juice," or "extract of limes" of the hair-dressers has much the same effect. Another thing very frequently applied is Tannin, and Glycerine of Tannic Acid has an undoubted effect on the scurf of the head. Its exact mode of action is not easy to explain. At the same time I think even that is to be regarded as of only temporary utility. I believe the only thing which permanently succeeds in stopping this kind of affection is, broadly speaking, absolute disinfection or sterilization of the scalp. It is a fact familiar to surgeons that wounds of, or operations on the scalp have always been regarded as more dangerous, more liable to give rise to unhealthy suppuration and pyæmia, than those of other parts of the body, the reason being that this part of the surface is swarming with micrococci of the kind which produce suppuration. This fact is an indication that you are not likely to make any condition of the scalp well without thorough disinfection. Speaking to an audience of qualified graduates, it might be enough to lay down

this principle and leave the details to you; but I will give a short outline of the mode of procedure.

The first thing to do is to treat the scalp thoroughly for a few, say three days, with Perchloride Solution, 1 in 1,000, or, if the skin is a delicate one, 1 in 2,000; or, use one of the antiseptic soaps, some of which are really efficient. But this treatment is not enough. Although the condition appears to be improved, it is apt to return, and irritation is often produced. Something seems to be required to improve the nutrition of the scalp as well as to destroy superficially the germs that may be there. The next remedy to apply is one undoubtedly useful in all sebaceous affections, namely, Sulphur, which has a most definite effect. Considering that we also want an antiseptic, what I generally prescribe with most effect is an ointment containing both Sulphur and Carbolic Acid:—

Sulph. Precip.	gr.xv
Ac. Carbol.	℥xv
Vaseline (or some excipient)	3j

to which you may add either two or three drops of Oil of Lavender or Oil of Bergamot or any other scent. Coal Tar solution or Creosote may be substituted, if preferred, for the Carbolic Acid. When this has been used for a week or two the effect on the scalp is extraordinary. The scurf disappears almost entirely, and the condition of the hair is improved.

Washing and pure antiseptic treatment should be repeated from time to time, say once a week. At the same time, brushes, combs, hat-linings, etc., should also receive antiseptic treatment, or be destroyed. After a course of treatment such as this, it will generally be desirable to restore to the hair some of the oily matter which it has lost. This may be done by rubbing in oil, or, still better, a soft pomade made with beef-marrow. At the present time, however, most patients object to any kind of greasy application.

For the seborrhœa or secondary acne of the face ointments are less convenient than lotions. For these we generally use the lotion often employed or acne, as follows:—

Sulph. Precip.	gr.xxx
Glycerini	3ss
Spir. Camphor.	℥ij-v
Liq. Calcis	3ss
Aqua Ros.	ad	3j

The amount of lime water must be less for

delicate skins. If you put this solution on the head, in the cases of which I am speaking, it is very likely, at first, to make the hair fall out. It is, therefore, a dubious application for the scalp. The old remedy of Perchloride of Mercury in almond emulsion (one grain or half a grain to the ounce) is also very useful on the face. Speaking broadly, the principle is antiseptics. As there is always, no doubt, also a certain amount of inflammation, besides the pure antiseptics it is desirable to use such applications as Coal Tar in the form of ointment or lotion or any similar preparations intended to cure chronic eczema. The treatment of eczema of the scalp I cannot here enter upon. Cases of seborrhœa of the trunk and other parts are also best treated generally in the same way; but a good deal of soap is an important part of the treatment. Sometimes, though not by any means always, this is enough by itself, if the woollen undergarments are carefully looked after. The ointment above mentioned may be used, or some mild mercurial, such as dilute Citrine Ointment.

We are often asked whether any rules are necessary for the general health or diet. I do not think the condition depends on the general health; but when there is any tendency to suppuration I have not the least doubt that, especially in children, the amount of sugar, and things capable of being converted into sugar, ought to be limited. In children these pustular spots, etc., are decidedly favoured by an excessive consumption of sugar in any form. In adults beer has a similar effect. But the discussion of this question belongs to the treatment of acne.

It is also true that in many of these conditions of the hair and skin there is bad nutrition generally, especially of the skin. There may be no real illness, but the skin looks badly nourished, and rather coarse. In all such conditions, independent of anything which can be called disease, Arsenic is very useful. It improves the complexion even of people suffering from no disease at all; and it improves the nutrition of the skin in cachectic or anæmic conditions.

I have said, also, the tendency to boils is often a marked accompaniment of seborrhœa; and here I have found that the only effectual treatment is thorough antiseptics of the skin. I have tried, as everyone has done, innumerable ways of influencing boils by diet, by alkalies or acids, and by other kinds of internal treatment, but with dis-

appointing results. Antisepsis does not cure the boils that are already there, but it prevents the formation of new ones, even though the patient has gout or other internal conditions supposed to favour them. At the same time I do not deny the influence of saccharine or rich diet as a predisposing cause.

With regard to seborrhœic eczema, I regard it as being a sort of combination of seborrhœic disturbance with inflammation; that is to say, an extension of the seborrhœa. I ought, however, to say that Unna regards the whole process from the beginning as being a kind of eczema. When the eczema is established it may usually be treated by the ordinary means; but if an eczema which is connected with a scurfy head is at all obstinate, one practical rule is always to try the effect of Sulphur, because Sulphur has an effect on the original seborrhœic lesions and also on the secondary lesions. It is recognized by the school of Unna that in all these conditions connected with seborrhœa of the head Sulphur is a great addition to other local treatment; and this I can fully confirm.

A LECTURE

ON

SOME OF THE COMPLICATIONS and SEQUELÆ of ENTERIC FEVER.

Delivered at Charing Cross Hospital, Nov. 18, 1893,

BY

JOHN ABERCROMBIE, M.D., F.R.C.P.,

GENTLEMEN,—The complications, and sequelæ of enteric fever, are, perhaps, more important than of any other malady; they are so numerous and so varied, and they have such very great significance with regard to the question of prognosis, the making of which on sufficiently reliable grounds, is, after all, one of the chief duties of a doctor, that it would seem well worth while to devote some time to their special consideration.

First, a few points with regard to the *temperature*. On a former occasion I mentioned that all cases in which the temperature became normal before the twentieth day, might be regarded as short, and all cases in which the temperature remained up beyond the twentieth day, might be regarded as long.

How short a case of enteric fever may be it is difficult to say. We have a patient now in one of the wards in whom the temperature came down to normal by the fourteenth day, or certainly the sixteenth; and I think I have seen the fever come to an end even earlier than that. Such cases are, of course, exceptionally mild; but one must remember that they have to be treated in exactly the same way as the most severe cases. Such cases are just as liable to complications and relapses as severe cases.

Where the temperature does not come down to normal by the twentieth day, and you have, therefore, a long case to deal with, the course of the temperature is no longer characteristic or typical, *i.e.*, the morning temperature is not by any means necessarily lower than the evening temperature, as it is all through the course of mild attacks.

The main causes of the prolongation of fever beyond the usual duration are two.

(1) In the cases in which there are no very grave symptoms, and you can find nothing to account for the fever, you may assume that the lesion has spread further up the bowel, and fresh Peyer's patches have become involved; in fact, your patient is getting a relapse before he or she has recovered from the original attack, this is what is termed a *recrudescence*. That, I think, is generally the case where there are no particular symptoms, and where the patient is perhaps not very seriously ill. That may be the case with a patient now in the Victoria Ward, a young woman who has had a very well characterized attack of enteric fever of considerable severity in some respects. She has now got to at least the twenty-fourth day, and her temperature is still very high, although she has no special abdominal symptoms now. There are no complications to which we can fairly ascribe the fever, and so we may assume, I think, that fresh Peyer's patches have become involved.*

(2) The other cause of continuance of the fever, is a spreading of the ulcerative lesion in the ulcers themselves, the ulcers becoming larger, spreading at their margins and eating deeper. That is, of course, a matter involving great danger to life, inasmuch as there is increased risk of hæmorrhage, increased risk of peritonitis, and increased risk of perforation; so that in cases where there were grave abdominal symptoms—a great deal of pain in the

* This surmise was verified a few days later by the appearance of an abundant crop of characteristic rose spots.

abdomen, a good deal of distension, and, perhaps though not necessarily, diarrhoea—if the fever continued one might infer very fairly that extension of the ulceration was taking place; and if we did arrive at this conclusion, it would make us be very guarded indeed as to what we said about the probabilities of recovery.

These are the two main causes, I think, apart from any complication, of the continuance of the fever. But it more often happens that the temperature does come down to normal by the twenty-first day; and then what may and does happen in a good many cases of which we have had instances in the wards lately, is that in a few days the temperature rises again. It is by no means uncommon, I think, for a person who has had an attack of enteric fever to get, during convalescence, a single rise of temperature for one day, what is called "an ephemeral fever," and that is almost invariably associated with some bowel trouble, generally constipation, and accompanied by a certain amount of pain in the abdomen. If you compare the temperature chart with the record of the action of the bowels, you will find that the ephemeral rise of temperature is almost invariably due to constipation, and when this is relieved, e.g., by Glycerine enemata, the temperature comes down again, and you have no further trouble. But that would not constitute a relapse. A typical relapse is practically a little attack of enteric fever; you may get all the phenomena over again, including a further development of spots, but it would not by any means be necessary to have a fresh crop of spots; all relapses do not present such a complete reproduction of the original symptoms.

A relapse may occur any time within ten days after the first attack has come to an end. It generally, I think, comes on pretty early, very often in four or five days. There is a young man now in the Alexandra Ward who has just passed through a mild relapse which commenced about five days after the termination of his fever; he came in here a day before his primary attack ended, but his temperature rose again, and he went through a mild relapse. I do not think he had any further development of spots. For this relapse we could find no cause; there appeared to have been no indiscretion in diet in his case. In the case of a child in the Alfred Ward three weeks ago, there was a very close association of the relapse with a sweetmeat given him by a boy in the next bed. He had come in on the very last day of his

primary attack; his temperature went down on the day of his admission; three days later, the day after he was given the sweetmeat, his temperature went up and he had quite a sharp relapse, the temperature going pretty high. If I were to judge from my own experience alone, I should be inclined to think that relapses were commoner in children than adults. That may not be so; but certainly I have had a considerable experience of relapses in children. One explanation may be that enteric fever is more apt to be overlooked in children than in adults. I have seen a good many cases come into hospital, not in the least diagnosed outside, and at such a late stage of the illness that the diagnosis of enteric fever did not suggest itself. A child would come in and have a little fever for three or four days, and then become apparently well; but in about a week or ten days when the diet was improved, we should find the temperature going up, and in the course of three or four days it would become evident that we had to deal with a relapse of enteric fever.

The relapse never lasts the twenty days, about ten days would be a long time for a relapse. Very often it does not last more than five or six days. In a few cases you may get a second or even a third relapse occurring with about the same intervals between each. I have in my mind a case where three such relapses occurred, of course prolonging the duration of the patient's illness.

I mentioned on a former occasion that a sudden fall of temperature denoted the probability of hæmorrhage from the bowel. Of course you may get a case where the fall of temperature is, as it were, the first intimation of what had already taken place, but where the blood has not been passed. It might also be consequent upon collapse from perforation.

On the other hand, an excessive rise of temperature—hyperpyrexia—may be the result simply of the severity of the attack. It is not usual to consider the fever in a case to amount to hyperpyrexia unless the temperature has reached 106° , although anything over 105° naturally denotes a very severe case.

We may next consider the *complications that have to do with the digestive system.*

Sore Throat is an extremely common thing in the early stages of enteric fever, and very often it puts one off the diagnosis a little. The patient complains chiefly of headache and feverishness, but also of his throat; and on looking at it you may

find it a little red, and with perhaps some exudation upon the tonsils. It is of no importance whatever; it is part of the general febrile condition.

In the early stages it is by no means uncommon to get *vomiting*. Generally that ceases when you succeed in regulating the diet, and finding what the patient can keep on his stomach. In the early stages it is a symptom of no importance whatever as regards prognosis. You need not be in the least anxious because the patient is vomiting; but in the later stages vomiting is, of course, a symptom of very different import indeed. In the third week, for instance, vomiting would probably mean the supervention of peritonitis, a complication we greatly dread. It happened once in my experience, and once only, I am glad to say, to see vomiting of blood: and I must confess that for a moment the practitioner who was attending the case and I were extremely alarmed. We did not succeed in ascertaining the cause at all; we could not even surmise its reason. The case was that of a gentleman nearly 60 years of age, who was having a moderate attack of enteric fever, when, about the twelfth day, he complained very much of pain in the right hypochondrium just under his ribs. His pain was so severe as to make him perspire freely. We examined him most carefully to find any cause of the pain, but in vain. What suggested itself was some diaphragmatic pleurisy; but we could hear nothing at all that proved its existence. We put some mustard leaves on, and the next day he declared himself very much better. On the succeeding day, however, about forty hours after the attack of pain he began vomiting altered blood, two or three mouthfuls at a time. He did this eight or ten times, so that altogether he vomited up a tangible amount. He was, of course, very much frightened at this, and so was everybody about him. What the cause of it was we never found out. We gave him a large injection of morphine, after which the vomiting ceased; he went to sleep, and we had no further trouble from this source, and he made an excellent recovery without any other bad symptom. That the blood was closely associated with the attack of pain two days before I think there can be no doubt.

As regards *hæmorrhage from the bowel*, a very much dreaded complication or symptom (which ever you like to call it) in enteric fever, you might possibly get it in the early stages; but if you do find blood passed from the bowel in the first fortnight it would be only very slight in amount,

being due to a mere oozing from congested vessels in the neighbourhood of the Peyer's patches, and would be of no importance as regards prognosis: but after the commencement of the third week when the sloughs may have separated, leaving ulcers, then you may get hæmorrhage in great quantity. As I have said, it is sometimes betrayed by a fall of temperature, the blood having been poured out into the bowel in some quantity sufficient to reduce the temperature without being passed per anum. In such a case the patient would be very much blanched and his pulse weakened. Generally, however, the blood is passed either with fæcal matter or alone. It may be coagulated or not; in the latter case, of course, it would probably be in considerable quantity, and passed before it had time to coagulate.

The passage of blood by the bowel is always a very serious and alarming symptom; but one must not jump to the conclusion that one's patient will certainly die. I may be exceptionally fortunate in the cases I have seen, but that has not been my experience. I have only once seen hæmorrhage from the bowel in enteric fever the actual cause of death, and even that was perhaps not a fair case, heart disease being an element in it. It was that of a child of 11 years with mitral disease in an advanced form; and no doubt that gave her a very poor chance indeed of surviving a considerable loss of blood during an attack of enteric fever. That is the only case in which I have actually known the loss of blood to be the cause of death. I have seen—not very many, because happily it is not very common,—but I have seen several cases, at any rate, recover after the occurrence of tolerably profuse hæmorrhage.

Another thing, one must always be on the look out for, and of very great importance as regards prognosis, is *Distension of the Abdomen*,—the so-called *Meteorism*. It is one of the things one should pay attention to from the first; when you first see the patient, notice particularly whether the abdomen is distended or not. If you see the patient in, say, the middle of the second week, the ninth or tenth day, and there is a good deal of meteorism, you may depend upon it the case will be one of considerable severity. When the meteorism becomes very extreme, it is possible sometimes, there is peritonitis as well; it is difficult to say. The most extreme case I ever saw was in a boy of 13, in whom meteorism began early, about the ninth day. On the sixteenth day

it was extreme, the abdomen being greatly distended and tympanitic all over. It remained thus from the sixteenth to the twenty-eighth days without any alteration whatever. It was an extremely severe case; but, thanks, I suppose, to his youth, he eventually got quite well. We never could make up our minds whether he had peritonitis or not. Of course the tympanites itself would be the cause of a good deal of pain and tenderness in the abdomen.

We come now to *Peritonitis*.

Remember that peritonitis may own several causes in enteric fever, of different importance both in regard to frequency and danger.

First (putting them in the order of danger) comes *Perforation*. That certainly would set up an extremely dangerous form of peritonitis. Without getting any actual perforation, the ulcers may grow so deep that the *inflammation spreads through the serous coat*,—the only one left,—to the other side, and so set up peritonitis by direct contiguity.

Among other sources of peritonitis, not so common, but still not to be altogether forgotten as possible, is the *Rupture of Softened Mesenteric Glands*. In enteric fever the mesenteric glands get considerably enlarged and very soft, and occasionally they may rupture.

Another source—not common, certainly—would be the development of an *abscess* either *in the ovary* or *in the wall of the bladder*, or, as sometimes happens, *in the rectus abdominis*. I told you, in speaking of the post-mortem appearances on a former occasion, that waxy degeneration was often found in the rectus abdominis, and sometimes abscesses may follow in that situation.

There may be other minor causes of peritonitis; but, of course, the first two are by far the most important—perforation and the spreading of inflammation directly from one surface to the other.

How are we to know when our patient has got peritonitis? Well, we need not be on the look-out for peritonitis for the first fortnight; it is not at all likely, unless the case is one of exceptional severity, that peritonitis would develop so early; but if, in the third week, your patient begins to complain of severe pain in the abdomen, pain much more severe than hitherto and of a stabbing character, if that pain is localized to any particular spot, if there is extreme tenderness over that spot, and the muscles of the abdomen tend to become contracted over and around it, you may fear very much indeed that your patient is developing peritonitis.

If the peritonitis spreads you would expect that the patient would tend to lie with his knees drawn up, so as to protect the tender spot in the abdomen as much as possible; the pulse would tend to become small and thready, and the face to assume a peculiar drawn expression—not a very pleasant thing to see, because you know what it augurs. Then you may get vomiting and hiccough. Vomiting coming on after a patient had developed such pain as I have described, would justify one in entertaining very grave fears for the result.

Perforation, which I have mentioned as one of the causes of peritonitis, may come on any time after the commencement of the third week; but one would not expect that the ulcers would be deep enough to lead to perforation before that.

Cases where there have been severe diarrhoea, hæmorrhage, or abdominal pain, are those in which you might feel anxiety that the patient might get perforation; but you may get perforation where there has been no diarrhoea at all, and little or no abdominal pain throughout. It may occur in cases that have been up to the moment of perforation comparatively mild. Most of you have probably heard of the so-called “ambulatory” cases, where people walk about, perhaps doing their ordinary work, until they are seized with severe pain in the abdomen which quite paralyses them, and renders them incapable of doing anything. They are carried into hospital in a collapsed state and die in a few hours, often before a satisfactory diagnosis can be made, except, perhaps, that of peritonitis. On post-mortem examination, you find the patient has had enteric fever; there is ulceration of Peyer’s patches, and one of the ulcers has perforated. I have seen one case of that kind. They are not very common; but they are well known, and many cases have been recorded: so that you must never forget that even in an apparently mild case you may get perforation.

The exciting causes of perforation generally are indiscretions of diet. I have known, for instance, a mother to give an orange to her child surreptitiously; and the child dying subsequently, orange pips were found in the abdominal cavity. One case I remember very well. It was particularly impressed on my mind by the fact that notwithstanding the death of the first child from this cause, when, three weeks later, the mother had another child in the hospital suffering from enteric fever, she was actually detected bringing oranges to that one. Whether she brought them with

homicidal intent or not, one could not say; but certainly it was suspicious.

A sudden collapse with all the attendant symptoms of collapse,—a feeling of faintness, and possibly, if the patient is able to speak, complaint of a sensation as of sinking through the bed, copious perspiration, lips of bluish colour with perhaps feeling of great cold and intense pain at a particular spot in the abdomen,—would make one very much fear that the patient was the subject of perforation.

If you examine the abdomen (which you would have to do with the very greatest gentleness) there is one physical sign which, if you find it present, would strongly confirm the idea of perforation having occurred, namely, that the abdomen has suddenly become fuller than it was, meteorism has become much more marked and the liver dulness is absent. If you were quite certain that these changes had taken place suddenly in the abdomen, you might be sure that perforation had occurred. There must be the presence of gas free in the abdominal cavity to explain such a condition. Even when perforation has occurred recovery is not absolutely impossible. One is obliged to say that a little guardedly because there is one condition extremely difficult to distinguish from perforation, and that is the so-called "pseudo-perforation" which results from the rupture of a softened mesenteric gland. One can readily understand that if a softened mesenteric gland did rupture it would set up very nearly all the ordinary symptoms of perforation. There would be profound collapse, involving a pinched expression of face, perspiration, cold extremities, and so on. Diagnosis between the two is well-nigh impossible, I think, and I do not know that it is of very much importance to make it. There would be no difference in regard to treatment in the two conditions, but it just enables you to hold out a faint hope when these symptoms have supervened that recovery may take place. I speak the more confidently on this point, because I have seen a case in which, certainly at the time it happened, we all agreed that perforation had taken place. The patient was a nurse, over 40, who had a very severe attack of enteric fever, and was not going on well. One morning she suddenly complained of intense pain in the abdomen, and became very much collapsed, blue, cold, and pinched, with very thready pulse; yet she ultimately recovered: then we thought that perhaps after all it was a

case of rupture of softened mesenteric glands. It does not matter which, however; she got well; that was some fourteen years ago, and she is still quite well.

As regards the complications referable to the nervous system, *Delirium* does not deserve to be called a complication; it is almost the rule in all cases of even moderate severity that, at night at any rate, there should be some delirium; but if the patient were delirious night and day, and remained so for a week, or ten days, or even a fortnight, or longer, you might regard it as a complication. If the delirium were so severe as that, you would have the usual concomitants of delirium, the sub-sultus tendinum, and the condition of restless moving of the fingers, plucking of the bedclothes, etc., known as carphology or floccitatio. It has been somewhat remarkable that the young woman in the Victoria Ward, now in a stage of recrudescence, has at no time had any delirium, though the case has been one of some severity.

Other complications referable to the nervous system may occur, and are worth noting.

First, I would speak of *Rigors*. Perhaps none of you saw a patient in here just two years ago with a severe attack of enteric fever. She was a young woman, 22 years of age, and was admitted on the 26th October, the fifteenth day of the malady. The case lasted a long time, owing to certain complications. On the 26th and 28th of December, more than two months after admission, she had rigors, the temperature on these two days going up to 104° and 105°. Yet absolutely nothing happened; she was not in any way the worse at the time or afterwards. The rigors did not seem to interfere with her progress or recovery.

I have seen rigors in several other cases which I shall briefly mention. The first patient was a young woman, æt. 26, who had a rigor on the twenty-eighth day of the fever, when the temperature went up to 106°; but nothing happened. She had no bad symptom afterwards. The case was a very severe one, but she was absolutely none the worse for the rigor. Another patient, a married woman, æt. 25, had two rigors, without any attendant bad symptoms. Another, a woman again, æt. 30, had a rigor on the twenty-second day. In her, certainly, the rigor was associated with constipation. In one of the other cases almost certainly, and possibly in the others, there was constipation also. It is noteworthy that all the patients in whom rigors have occurred, have been women, and they have occurred

rather late in the malady. It is quite clear, I think, from these cases alone, that a rigor may occur without necessarily meaning anything very serious. Indeed that was long ago pointed out by Dr. Gee in a paper in the St. Bartholomew's Hospital Reports, in which he instances two cases, both women, in whom rigors had occurred without any serious import, the patients having got well.

But you must not go away with the idea that a rigor is always perfectly harmless. It may mean peritonitis. That should always come to one's mind first, viz., that a patient who has just had a rigor may be developing peritonitis. That certainly was the case in one instance where a woman, æt. 42, had a rigor on the twenty-sixth day of her illness, just after an action of the bowels. The rigor lasted twenty minutes. She had some suggestions of peritonitis before; and after that the symptoms of peritonitis rapidly developed themselves, and she died in about six days.

(To be concluded).

THERAPEUTICAL NOTES AND FORMULÆ.

Treatment of Typhoid.—Dr. Eliot draws attention to the value of rest in the treatment of typhoid, and emphasizes once again the imperative necessity of not allowing *any* patient with pyrexia to remain out of bed unless enterica has been definitely excluded as a possible diagnosis. If enterica be diagnosed then the rest must be *absolute*; the patient must not leave his bed under any pretence whatever. He praises the use of rather large doses of Calomel; if the case is seen in the first week four doses of gr.vij to gr.x are to be given at intervals of twenty-four to forty-eight hours; if griping be caused a small dose of Opium is to be added, and if the Calomel does not open the bowels then he advises the additional use of ʒj doses of Sulphate of Magnesium. If seen after the middle of the second week the Calomel treatment must be omitted. For specific drug treatment he advises the following:

R. Acid. Carbol. ... ʒj
Tr. Iodi ... ʒiij

M. Ft. mist. Sig.: M̄iv in a wineglassful of water every four hours.

To combat nervous symptoms, and especially insomnia, he prefers Bromide of Sodium to any other single drug. Stimulants are only required when the heart shows distinct evidence of flagging.

New York Med. Rec., 21, 1893.

Tapeworm.—Chloroform greatly increases the efficacy of Male Fern. Twenty to fifty drops may be given.—*Duhomeau*.

For Granular Conjunctivitis:—

R. Hydrarg. Oxyd. Flav. ... gr.ijj
Zinci Oxyd.
Thymol... ... āā gr.iss
Camphor ... gr.ss
Cocain. Mur. ... gr.iss
Vasalin... ... ʒj
M. Ft. unguent. Signa: "Eyesalve."

REVIEW.

Diseases of the Skin. By MALCOLM MORRIS.
(Cassell & Co., Limited.)

Published at 10s. 6d.

Messrs. Cassell's manuals have deservedly gained for themselves a very high reputation amongst students and practitioners, and we can honestly say that Mr. Morris's book is very likely to sustain that reputation. The coloured plates at the commencement are excellent specimens of the art, and reflect great credit on Mr. Burgess, though there is the unavoidable disappointment in expecting too much from pictures. Caps. I. and II. on the pathology of the skin, and classification of its diseases respectively, are very well done; but Cap. III. on the principles of diagnosis, seemed to us more especially excellent: it is short, but very much to the point, and will be very useful if thoughtfully studied.

The next five chapters are devoted to "affections of the skin, dependent on nerve disorders;" it would be easy to quarrel with Mr. Morris's grouping of purpura and peliosis rheumatica, along with pemphigus and scleroderma. But we feel little disposition to do so, by reason of the excellent manner in which he has treated the individual diseases. Eczema, pityriasis, and psoriasis are well dealt with next; parasitic affections, in the widest acceptance of the term, occupy a large space, and then diseases of the appendages, and the whole is concluded by new growths and malformations. Prescriptions are scattered with profusion throughout the work, many of which are old familiar friends of well-tried virtue. We heartily approve of Mr. Morris's definition of eczema, though it may not be all-sufficient, and we note he uses the term catarrhal in a very different sense to that which Mr. Hutchinson gives it.

While thus giving unstinted praise to the book as a whole, we feel compelled to draw attention to one or two weak spots which we hope may be strengthened in a future edition: thus on p. 11, a tubercle is defined as a solid elevation of the skin, larger than a pea. We must enter an emphatic protest against such a use of the word. Nodule and nodular are quite equal to the representation of the object here intended, while tubercle and tubercular have long had a specific meaning attached to them in association with a bacillus, and to this connection we maintain they should be exclusively devoted. Sclerema neonatorum is described as though it were always universally distributed over the skin of its victims: this has certainly not been so in the few cases we have had the opportunity of seeing, though they were well-marked examples of the affection.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 27, 1893.

A LECTURE ON SOME OF THE COMPLICATIONS and SEQUELÆ of ENTERIC FEVER.

Delivered at Charing Cross Hospital, Nov. 18, 1893,

BY

JOHN ABEROROMBIE, M.D., F.R.C.P.

(Concluded from p. 128.)

In one case we have seen, as a complication, *Mania*. That was the patient to whose case I have just alluded, who was in here two years ago, and who had the two rigors so very late in her malady. When she first came in (on the fifteenth day of her illness) it was a severe case, and she was very delirious at night; but very soon we found she was wandering in her mind in the day time also, and after a few days it became clear she was suffering from something more than delirium. She turned out to be quite maniacal. She had delusions of sight and hearing; she thought she saw people coming to take her away, and fancied she heard noises. One day when she was not so carefully watched as she might have been she succeeded in undoing some of the sacking under the bed she was lying on, and getting a piece of rope with which she tried to strangle herself. She got quite well, both as regards her enteric fever and her mental symptoms. I am sorry to say, however, that about a year afterwards she began again to have some hallucinations, so that I very much fear that her insanity may have become more permanent.

Another complication, which, happily, I have only once come across, but which at the time was a very alarming one, is *convulsions*.

In nearly all the recorded cases of convulsions, these have been associated with much albumen in the urine and speedily followed by death.

The case I have to relate under this head was one of extreme severity in a married lady of 21. The enteric fever came on shortly after her confinement; characteristic spots were recognized just three weeks after that event. It was an extremely severe case from the first; and on the evenings of two days, the fifteenth and seventeenth, she had

convulsions, two on each day. There was some albumen in the urine, although not more than would be accounted for by the severity and height of the fever. Of course the occurrence of convulsions was extremely alarming, and there was nothing we could make out to account for them. She was taking her food exceedingly badly, and was rather troublesome about it; but the bowels were acting sufficiently,—about every other day. There was no diarrhoea, the motions being semi-solid and pultaceous. When the second batch of convulsions occurred it suggested itself to us that there could, at any rate, be no harm in giving her an enema. In children convulsions are very often due to intestinal irritation; this patient was young and extremely exhausted by the circumstances under which the attack of enteric fever occurred, so that we thought it possible that she was really reduced to the same condition as a child, and that the convulsions might be purely reflex, and, possibly, due to intestinal irritation. Although the bowels had acted sufficiently the day before we gave her an enema, and with the best results. It removed a very large quantity of semi-pultaceous, offensive faecal matter,—a much greater quantity than one would have supposed possible after the action of the bowels the day before. We had no more trouble from the convulsions, and our patient, after some vicissitudes made a complete recovery.

One other point with regard to the nervous system I should like to mention, though it is rather of the nature of a sequela than a complication, and that is a condition of *imbecility*. Some of you may possibly remember the case of a youth of 18 or 19, in here some two years ago, about whom all the history we could get when he was brought in was that he had been suffering from "brain fever." He had been quite well, it was said, up to the time when the "brain fever" came on. On admission he certainly appeared to be quite imbecile. He was extremely feeble in body: he could hardly stand, and when he sat down it was "all of a heap," the muscles of his back being very weak; his face was perfectly expressionless, and such as you might see in a severe case of chorea when the movements happened to be in abeyance. He reminded me very much of a child I had seen years before, who presented exactly

the same appearance of hopeless imbecility, and who had turned out to be merely suffering from the debility consequent on enteric fever. I expressed an opinion then that probably his "brain fever" had been really enteric fever, and when his mother was questioned, she said, "If you want to know my opinion I always thought all through it was 'typho' fever; but the doctor thought it was brain fever." Our patient in the course of three weeks got perfectly well, and became quite rational, which fairly confirmed the idea that his original malady had been enteric fever.

My experience of these two cases leads me to think that the condition of such patients is due to their having taken a wholly inadequate supply of nourishment during the attack, because one never sees it as a sequela of cases treated in hospital. The question of nourishment is of extreme importance; and in cases treated without the supervision of good nurses it is probable that this point does not get the attention paid to it that it deserves and requires.

Perhaps the chief thing with regard to the circulation, apart from heart failure, of which I spoke when dealing with the symptoms, is that sequela which is by no means uncommon, viz., *thrombosis of the femoral vein*. It may come on any time during convalescence; I cannot exactly say when, as we do not very often see it come on in patients we have actually under our care in hospital; but very often patients come in with thrombosis of the femoral vein after an attack of enteric fever treated at home. It would always be well to warn your patient (who will recover from the thrombosis in due course, though it may be a question of months before the swelling goes) that after walking or other exertion the affected leg will, for a long time, perhaps for two or three years, perhaps for the rest of life, be liable to a return of the swelling.

With regard to the *respiratory system* :—

Bronchitis is rather common. A slight degree of bronchitis would hardly amount to a complication; but *pneumonia* and *pleurisy* would be complications. There is nothing special about their course in regard to this malady, but of course their occurrence adds to the gravity of the prognosis.

The two special respiratory troubles of enteric fever have reference to the larynx.

There is a special form of *ulceration of the larynx* which is more or less peculiar to enteric fever. It used indeed to be thought that the ulcer was exactly analogous to the ulcer in the Peyer's

patches. I do not know that that was quite true; but, at any rate, there is a special liability in these cases to ulceration in the larynx which may lead to *perichondritis*, and sometimes to *necrosis of the thyroid cartilage*. The occurrence of laryngeal symptoms during the later stages or during convalescence should make us think of these complications.

As regards other sequelæ, you will occasionally see, after severe cases of enteric fever, the patient get a crop of *boils*, or possibly a number of *abscesses*, either subcutaneous, or, sometimes, subperiosteal. These would require to be treated on general principles; and are to be regarded as a sort of pyæmia. In one case, that of a boy of 16, I remember to have seen a *joint lesion*; he had suppuration in his elbow joint, and, I suppose, a certain amount of permanent damage.

A CLINICAL LECTURE

ON

DISLOCATION OF THE FEMUR.

Delivered at University College Hospital, Nov. 27, 1893,

By CHRISTOPHER HEATH, F.R.C.S.,

Surgeon to the Hospital.

You saw last week, Gentlemen, a case of dislocation of the femur. Now, these cases are not, of course, very common; but it happens that we have had a good many during the last twenty years. I have taken the trouble to run through the reports of this hospital for that period, and I find that during that time we have had seventeen cases. We had one in 1871 (I need not trouble you with the details); two in 1872; one in 1873; one in 1876; two in 1879,—and one of these was a supra-pubic dislocation, which, of course, is the rarest form; two in 1878; three in 1880; one in 1883; one in 1885; one in 1887; one in 1888; one in 1889; and none in 1890. It is rather remarkable that the last four cases were all in young persons. In 1885 there was a child of 12 years only; in 1887 the child was 11½; in 1888 the child was but 6; in 1889 only 5. You may take it from me that these were *bona-fide* dislocations, the result of accident, and not the form of dislocation which we call pathological. Of course, we are all familiar with cases of hip disease, where the capsule softens and the head

of the bone slips out; and these we term "pathological dislocations;" but I am not speaking of that subject to-day. All the cases I am going to mention to-day are cases resulting from accident.

The man now in the ward is 33 years of age, and a labourer. Fifteen weeks before his admission here he had an accident. In jumping off a two-wheeled cart, which he had been driving, and on which he had been sitting in front with his feet upon the shaft, his foot caught in the horse's nosebag, which was swinging from the shaft, and he fell to the ground,—in what position, however, he does not know. He still held the reins, and stopped the horse. He says the wheel ran up his leg, but did not run over him; but he is not at all clear on the point. He was taken to a doctor within ten minutes of the accident, and the treatment employed consisted in rest, liniments, and so on. About six weeks afterwards he got out of bed, using first a pair of crutches and later on two sticks, and began to get about pretty fairly. But then, I suppose, the doctor's conscience smote him, and he thought he had better, perhaps, send the man to the hospital. When he arrived here there could not be a doubt, I should say, to even the meanest capacity, that it was a *bona-fide* dislocation of the femur on to the dorsum ilii. The next day I sent the man down for the F.R.C.S. examination, and on the following day, the Wednesday, I had him photographed. Here you see the result. The front view shows you very well the deformity. You will see that the left limb is inverted, that it is shortened, and that the ball of the great toe rests on the instep or thereabouts. The inversion of the limb is undoubted. If anything, there is a little widening of the pelvis, the head being on the dorsum ilii, and not in the sciatic notch, the prominence of the trochanter is a little exaggerated. If you have a case where the head of the bone has dropped into the sciatic notch, then you sometimes get remarkable narrowing of the pelvis. I had a side view also taken; but I am rather disappointed with that; it does not show the deformity caused both by the trochanter and the head of the bone as well as it should do.

How is it this dislocation was overlooked? I really cannot explain it. I tried in every way to get from the man that the parts were a good deal swollen before the doctor saw him; but it was not the fact. It is evident, from the notes, that the medical man saw him ten minutes after

the accident; there could not, therefore, be much swelling; and it is obvious that it is one of those cases which have been overlooked, and which from time to time we meet with. We all make mistakes, and therefore we ought all to be charitable to others. I have purposely never inquired who the gentleman was that saw the case.

A word or two about the frequency of this dislocation. Malgaigne gives 491 cases of dislocation in the Hotel Dieu, the great hospital in Paris. Of these dislocations 321 were of the shoulder, and only 34 of the hip; but the hip was the next most frequent to the shoulder. Mr. Henry Morris in his article in Holmes's System of Surgery gives the statistics of Middlesex Hospital for ten years, and I may say, that these include both in- and out-patients. The statistics of this hospital, as given in our report, only include in-patients; but I take it that there are very few cases of dislocation of the femur among the out-patients. Dislocation of the shoulder is, of course, commonly enough treated in the out-patient room; but all cases of dislocation of the femur are taken in as a matter of course. At the Middlesex Hospital, where both kinds of patients are taken together in the statistics, there were in ten years 283 dislocations, of which 133 were of the shoulder, and only eight of the hip; so that, you see, the proportion is very much the same in different hospitals.

As to the method by which a dislocation is produced there has been a great deal of discussion. I do not know that the point is absolutely settled yet. Mr. Morris, the gentleman I have before alluded to, wrote a paper in the Medico-Chirurgical Transactions of the year 1878, in which he discussed this question at some length, and gave the result of some experimental inquiries which he had made. I give you shortly the conclusions he came to. The capsule of the hip joint, he said, is, as we know, weakest behind, and, possibly, below; and he particularly insisted upon the fact that the perforation was always at the *lower part behind*; that the bone then slipped up upon the dorsum ilii; and that therefore a dislocation on to the dorsum ilii is what is called a secondary dislocation. That is a view which has been a good deal discussed; and Mr. Eve, who was at that time Registrar at St. Bartholomew's Hospital, three years afterwards published a case, and contradicted Mr. Morris. It is the pleasant way we have, that one surgeon always contradicts another.

The value of Mr. Eve's paper particularly is that he gives drawings from actual specimens which I have here, and which even at a distance I think you can see. Of course in these cases the patients either died at the time or soon after the injury, and opportunity has been taken to make the dissection. We have also here from the museum of University College a specimen of which I do not know the history, as it did not occur in my wards. In it you see the head of the bone; the quadratus femoris; the obturator externus torn, and the obturator internus stretched over the head. It is a dislocation, therefore, "below the tendon." If all the dislocations take place, according to Mr. Morris's suggestion, through the lower part of the capsule it seems odd that so many of them should find their way afterwards up to the dorsum ilii; but the fact is that there are undoubtedly many cases—and I believe our patient's case was one of them—where the dislocation takes place directly backwards through the posterior part of the capsule, of course, but *through its upper part*, tears through some fibres, no doubt, of the gluteus medius and minimus, and comes out "above the tendon," as Bigelow would call it.

This, then, was a case of dorsal dislocation, or dislocation above the tendon; and it is my belief that it was a direct dislocation. Of course, fortunately, we have had no opportunity of a post-mortem examination, and we shall not have one. But, Mr. Morris would argue, True it was a dorsal dislocation; but first it was a dislocation backwards below the tendon, and then, as a result of the man walking about, and so on, the head of the bone was pushed up, and it became a dorsal dislocation. Well, let it be how it may, there is no question that there are two dorsal dislocations which we have to recognize. One is that in which the limb is inverted and very considerably shortened, the toes rest on the instep, and, the trochanter being more prominent than usual, the head of the bone can be felt on the dorsum ilii, as in this patient. This is what Sir Astley Cooper called the *dislocation upwards*, or the *dislocation on the dorsum ilii*. It is now the fashion to call it *the dislocation above the obturator tendon*; but it is the same thing. In addition we have the second form of backward dislocation, where the limb is not much shortened, where the great toe rests against the opposite great toe: where the limb is more inverted but not so much flexed, and where the trochanter, instead of being prominent is apt to be

depressed; and therefore we have narrowing instead of widening of the pelvis. In addition, in the sciatic dislocation there is usually a good deal of pain down the limb from pressure on the sciatic nerve.

To finish the subject, although we have not a case at present, I would remind you of the other two forms of *forward* dislocation—forward and downwards into the obturator foramen, where the patient's limb is advanced and his body flexed; and, the rarest of all, the dislocation on to the pubes, where the limb is abducted and everted, and you feel the bone actually on the pubes close to the pectineus muscle.

We will go back for one moment to the anatomy of the hip-joint; and I have here a preparation to show the capsule. Here we have the posterior and lower part of the capsule, which is the thinner part; there we have the anterior part of the capsule, which is very much thicker; and you remember it has in front of it the great psoas and iliacus muscles supporting it; and it is this part on which Bigelow has laid so much stress, and which he has called by a separate name, viz., *the inverted Y-ligament*. I have Bigelow's book here, and will show you his own diagram, which no doubt is perfectly familiar to you all, as it has been copied into other books. It shows very prettily the two portions of the anterior part of the capsule, which is dignified by the name of the Y-ligament. We have in all anatomical books for years described an ilio-femoral ligament: and we have here another slip of it which really does not always exist. It is an exaggeration to say that in all cases you can make out a ligament like that, though you can in favourable subjects. However, the main point is this, that the anterior part of the capsular ligament is the strong part; the posterior part is the loose and weak part; and whenever a dislocation happens the head of the femur passes much more frequently through the posterior part; indeed some people go so far as to doubt if it ever comes through the front. Remember, too, that we have the ligamentum teres inside attached to the bottom of the acetabular cavity and to the head of the femur. Deepening the cavity we have the cotyloid ligament which maintains the head of the bone in the socket.

Now, How does the dislocation occur? (I am speaking now only of the dorsal dislocation.) Mr. Morris in the paper to which I have already referred is very strong about it that it always occurs

in the position of abduction of the thigh. He says that if you have the limb abducted, a very little force will drive the head of the bone through the lower and posterior part of the capsule (the weak part), and that then being once dislocated it may be pushed up to any point, either lodging in the sacro-sciatic foramen, or coming up on to the dorsum ilii. Now, I have seen a good many experiments made at different times on this point, and I must say I am not at all convinced of the truth of that statement. If in the dissecting room or post-mortem room you want to dislocate the femur, the simplest way is to cross the femur well over to the opposite side, which drives the head of the bone a little bit out, and then forcibly to jerk it, when out it comes. I have done this and seen it done many times, and I have not the slightest doubt that that is the way the accident generally happens. Think for a moment. When the limb is well adducted and crossed over the opposite limb, already the head of the bone is a little bit out of the socket. A violent jerk breaks the ligamentum teres, drives the head of the bone either through the capsule or tears away the capsule sufficiently to let it out, and there is the dislocation produced.

Now comes the question about reduction. This accident had happened fifteen weeks before the man came here. 105 days had elapsed,—in fact, the reduction took place on the 106th day. That is a long time for a bone to be out of its socket, and yet to be got back. But on looking up the literature of the subject I find there are cases of longer standing than that. There are cases on record—and I believe true ones—where even a year has elapsed and yet reduction has taken place. Finding that it was not quite a third of a year, I thought I might very well venture to try in this case, and we were successful. I think all surgeons in the present day are agreed that the manipulation treatment is better than the extension. Bigelow, who investigated the subject extensively, and whose book I have just shown you, has strong views upon it, and, I think, correct ones. His view is this: that what you want to do is to flex the limb and to adduct it with the object of relaxing, in the first place, the strong Y-ligament; then, having done that, that you should lift up the limb, bringing it upwards to a right angle with the abdomen, then circumducting it outwards you bring it down so as to get the head of the bone into the socket.

I had a good many difficulties to contend with

last Wednesday, one being that the limb was fixed in its new position. I did not know exactly what the condition of things might be, but evidently there was a beginning of something like a new joint; and I had, therefore, to work very hard at first to break down the adhesions, and get the limb to move at all. However, after a time, we were able to flex it and adduct it; then to abduct it and rotate outwards and bring the limb down. I repeated that two or three times, but I did not get the head of the bone in. Mr. Pollard helped me, and we were getting, apparently, no nearer success when, as the limb was everted, Mr. Pollard gave it a slight tilt upwards and the head of the bone slipped in, and is now in its proper position.

The theory of the manipulation treatment is that the ligament being the fulcrum and the femur the lever, you should adduct and flex and lift up; then circumduct outwards and bring the limb down parallel to its fellow; and if you are lucky the head of the bone will slip through the rent and into its place. Of course, after fifteen weeks, the rent must have somewhat repaired; and it is extremely probable, therefore, that I had more difficulty in that way because of the repair; but still the bone went in with such an audible snap and so cleanly, and remains so thoroughly in its place, that I feel sure there is no fracture, and that the head of the bone slipped through the capsule into its proper position.

In an interval of trying manipulation I thought I would give a demonstration how to apply extension; so, putting a wet bandage round the lower part of the thigh, and over that a skein of worsted made up into a clove-hitch, with my foot against the man's perinæum in order to get a fulcrum, I pulled, but did not produce the least effect. I hardly expected I should, but I wanted to show you how the thing could be done.

In former years I have reduced more than one recent dislocation of the femur in that way when the parts were all relaxed, and in a recent case you can pull well down and the bone goes in pretty easily. But I think the method of circumduction is better on every ground; still, it is right to warn you that accidents have happened with it. In an elderly patient the femur has broken, and that would be a very awkward complication. In this case I was careful what I was about. I felt a certain amount of giving of tissue; but that was merely the adhesions between the head of the

bone and the dorsum ilii. I was careful not to put on too much force, because I might have broken the femur, and, no doubt, as it was, I necessarily lacerated some of the muscles of the thigh, and possibly tore some of the ligamentous structures, too. However, "all's well that ends well." I am keeping the man in bed with a long splint on. I took it off just now and found everything in its place, and I have no doubt he will make a good recovery.

Just a word or two about the other form of *dislocation into the sciatic notch*. If it were a dislocation backwards below the tendon, the head of the bone would lie somewhere about the sciatic notch, although not necessarily actually in the notch. It comes back and presses upon the muscles which come through the great sciatic notch, or it may get impinged upon the spine of the ischium and lodge on the lesser sciatic notch. When it really gets into the sciatic notch there is flattening of the buttocks. Another diagnostic symptom, useful to remember, is that if you put your finger up the rectum you can feel the head of the bone actually in the sacro-sciatic notch. In doubtful cases, therefore, it is well to have recourse to that method. If you think it is a case of sacro-sciatic dislocation the method of reduction will be exactly the same: you would adduct, flex, lift up, abduct and rotate outwards, and then bring the limb down.

So much for these two kinds. Just one word, to complete the subject, about the other two forms of dislocation. I show you the diagram of the *dislocation into the obturator foramen*, with the limb advanced and the body somewhat flexed; and, lastly, that of *dislocation above the pubes*, with the limb everted and somewhat flexed. Now, in these two dislocations forward, of course the head of the bone will come somewhat in the position that is represented there, that is to say, it will slip past the Y-ligament and through the anterior part of the capsule. We had some years ago in Mr. Marshall's ward a woman who had fallen from the top of a house, and dislocated her femur. The limb was inverted, and attempts were made before admission to reduce the dislocation. (This is her account, at least.) She was not admitted here until the sixth day, at which time the limb was everted and two inches short. It was reduced. In 1880 we had a case, also a woman, who fell 60 feet. Her limb was everted, half-inch short, with the head of the bone on the pubes. The dislocation was

reduced by rotation inwards and flexion. I mention the two cases because they are of different kinds. Because the limb was everted it did not show that the earlier one was a dislocation on to the pubes. It was six days old and had been well pulled about. It was really a dorsal dislocation, in which the parts had been so torn by the treatment before she came in, that you could move the limb pretty well anywhere you liked. But the case in 1880 was a *bona-fide* dislocation on to the pubes, in a young woman of 23. I saw it with Mr. Marshall; and it is the only case of dislocation on to the pubes I have ever seen. It is so rare that one does not see above one or two in the course of a life-time. The reduction of these two dislocations is simple enough to remember. It is just the reverse of the previous method. In the case of the dorsal dislocations we circumduct outwards; in the reduction of the two forward dislocations we circumduct inwards. The patient is put on his back in exactly the same way, the limb is flexed, and then it is circumducted inwards. That rotation tends to bring the head of the bone downwards towards the acetabulum, and with a little traction it slips into its place.

The Y-ligament is equally important in both kinds, and it is very important that you should not tear that. I have known such force used that the ligament was torn through, in which case you get that curious condition in which you can put the limb in any position you please. It is an easy experiment to do on the dead body in a thin subject.

So much, Gentlemen, for this case. I think I have given you the principal points about it; and it just illustrates the importance of making a diagnosis immediately after an accident, and that even if a mistake is made at that time it is possible to repair the error later on. This man I have no doubt will get a perfect hip, his capsule will be quite sufficient to keep the bone in position; the ligamentum teres will not unite in all probability, and should we a great many years hence have the opportunity of examining the interior of the joint, it will be found that the parts are smoothed off there, but that no repair has taken place in the interior of the joint.

Bolls.— R Hydrarg. Perchlor. ... gr.ij
 Acid. Carbol. ... mxx
 Sp. Vini Rect. ... ℥iiss
 Aq. ... ad ʒij

M. Ft. lotio. Sig.: To be applied as a compress constantly.

SOME POINTS IN HEART DISEASE.

A Paper read before the South Eastern Branch of the British Medical Association at the Quarterly Meeting at Reigate.

By FRED. J. SMITH, M.D., M.R.O.P.,
Assistant-Physician to the London Hospital.

GENTLEMEN,—The medical journals, be they weekly, monthly, or quarterly in appearance, as well as our annual publications, teem with articles on new drugs and their scientific experimentation, with philosophical papers on the theoretical aspects of laboratory medicine and transcendental pathology, with deep disquisitions on the precise details of new instruments and their revelations, in fact, with so much theory of what disease, its investigation and its treatment ought to be, that my mind gets into a whirl, and I often think that I, and others with me, are in no inconsiderable danger of forgetting that we are clinicians first and scientists later, that the bedside and not the laboratory is our legitimate sphere. With this train of thought in my mind I deemed it better and possibly more interesting to you to bring forward a few practical and clinical notes on valvular disease of the heart, using statistics and science only so far as they throw light on the path of practice. In speaking thus, let me not, for one moment, be taken as despising or disparaging scientific medicine; no one is more keenly aware than myself of its absolute necessity if we are to progress in our art, and I yield to no one in my admiration of the accuracy, patience, and perseverance of our modern investigators; but on the other hand, no one knows better than myself the enormous amount of time, trouble and patience required to produce one little fact expressed perhaps in half a line of print.

Take rabies, tetanus, diphtheria, as examples of diseases investigated by the scientists, and by what figure can we represent medicine's gain from their labours—it is beyond price; still, as teachers and practitioners of the healing art, we have not the time for such deep and accurate laboratory work, so let us turn to the bedside.

A little while ago I was talking with a country practitioner, and I remarked that someone I knew could not tell with the stethoscope whether the aortic or pulmonary, the mitral or tricuspid valve was at fault, "Oh," said my friend (he was reckoned

a good man of his year, 1867, was at one of our largest schools, and now deservedly has a great reputation as a healer of the sick) "we never got so far as that in my time, we were taught to feel the pulse and note accurately the symptoms presented by the patient." That conversation made a deep impression on my mind, for it is now my constant experience in the out-patient department, that when I ask a student to examine a case with a valvular bruit, he will tell me with commendable accuracy the nature or character of the murmur, and the valve at which it originates, but if, away from the patient, I say yes, that is all right, but is the man suffering from heart disease or something else? he has to return to see if the beat is regular, to feel the pulse, to listen for crepitations in the bases of the lungs, in fact, so busy has he been with the valves and their supposed incapacity, that he has forgotten the organ that uses them, and passed by the patient who possesses them as unworthy of notice. Such experience helps one to understand my late honoured teacher, Dr. Sutton, when he says "It is of assistance in listening to the heart to be a little deaf." He meant, I take it, that the bruit was very little, but the rhythm of the heart and the general condition of the patient very much in dealing with a heart with diseased valves.

Let us look at the matter from a practical point of view, given a certain set of symptoms which might proceed, say, from heart, kidney or liver, etc., what are our objects? First, to determine, if possible, which is the organ primarily at fault, or if more than one; second, to judge whether the lesion from which the organ or organs are suffering is removable or not; third, to apply our therapeutic measures to the removal of the cause, or, failing this, to treat the symptoms purely and simply.

The first two objects come under the heading of diagnosis, and the question I would raise is this. Will the accurate identification by the stethoscope of the individual and combined bruits help us to the third object, which, for us, is the most important? Broadly speaking, I should answer in an emphatic negative. Lest I should be held to despise the teachings of our great cardiac specialists let me hasten to add that when no bruits are present the stethoscope is of the greatest value, both for diagnosis and prognosis, in estimating the finer degrees of alteration from the normal in the two cardiac sounds and in the interval which

should separate them, degrees of shortness in the first sound indicating failure, or clinical dilation of the heart, while degrees of length indicate hypertrophy, or nature's cure. But when bruits are present I persist in maintaining that the stethoscope is of much less use than an eye or a hand well trained in observation, for these additional or adventitious sounds will, as a rule, mask the natural sounds, the character of which still remains of so great importance

We must never forget that it is not the incompetency real or relative, nor the stenosis, which causes the symptoms, but the failure of the musculature of the organ, and this failure shows itself not in the bruits, but by signs and symptoms recognizable by other means than hearing. No, the stethoscope may help us in judging whether the heart is the prime offender, but it alone will certainly not settle the question for us. That I am not alone in this opinion of the lowly merits of the stethoscope in organic disease of the heart I am sure: certainly I know of one physician lately deceased who openly boasted that he never used that instrument; while his writings I consider are most useful guides to bedside treatment, and his success as a physician was attested by the crowds that flocked to his out-patient department from far and near. Ask yourselves, Gentlemen, do you withhold *Digitalis* or *Strophanthus*, etc., if you hear a mitral presystolic bruit, or give them with a systolic, do you alter your drugs if aortic bruits are present in addition? I think if you answer honestly you will say no; you alter your treatment with reference to the symptoms exhibited and the organs upon the assistance of which you rely in helping the heart. If it were then objected that certain symptoms arise from certain lesions to be diagnosed by the bruits, and that the diagnosis of the bruits is essential to understand the lesion, I should reply that such certainly in broad outline is true, that, for example, the symptoms of pure aortic lesions are generally localized to the brain and often associated with pain, while pure mitral lesions are usually general in their symptoms, and but infrequently associated with anginal pain; but none the less does it hold true that it is the symptoms before us, however anomalous, which claim our attention, and not some fanciful ideal in our own minds as to what ought to be present from the bruits we hear.

As a final argument before leaving diagnosis, let me quote a sentence from statistics I collected for my pamphlet on "Problems in Cardiac Pathology:"

"Interpreting these figures into clinical language (the figures were obtained from 545 autopsies with valvular lesions found and bruits heard before death) they mean—that out of every 100 hearts we listen to, in 36 the bruits will give us exact information; in 10 they will lead us absolutely astray; while in 54 they will give us information either in excess or defect of the truth. This can hardly be regarded as a masterpiece of scientific accuracy, and its lesson is certainly one that I have already hinted at, viz., to rely less on our mechanical aids to diagnosis and more on clinical observation."

I will now proceed to say a few words on prognosis in valvular disease. The subject presents itself in two very different aspects. Firstly, what influence on the duration of life has the presence of damaged valves? and, secondly, what are the prospects of recovery of each individual case of what I may call an acute attack of *morbus cordis*?

On the first aspect of the question I do not propose to say very much, as it is of secondary interest in everyday practice and statistical evidence is vitiated by the want of similarity of circumstances and of details in the individual cases; but there are two directions at least in which we may be brought to face the problem, so I will briefly mention some points for observation and criticism.

As a matter of life insurance are we justified in incontinently rejecting all persons with valvular organic bruits? Dr. Tidy remarks nothing is more uncertain than the exact duration of an individual life, but nothing is more certain than the average duration of many lives, provided that the average is obtained from sufficiently large numbers. This statement appears to me to indicate that we ought to be able to obtain data sufficient to enable us to classify lives where organic valvular mischief is present. In all cases it is obvious that some additional premium must be asked; for we must remember that from the moment a valve is permanently impaired in its efficiency, in whatever direction and in however slight a degree, a new standard of circulatory pressure and rate of working must be established—and that really an artificial one. Nature has provided us with organs of life on a very lavish scale, but even her resources are limited; and a heart in which compensation has been established is not in the same elastic condition altogether as one the valves of which are natural and the muscle untouched. Notwithstanding this, I certainly think that much might be said against an

absolute rule for rejecting all lives with organic bruits indiscriminately, as we must all know cases where persons thus suffering have lived to a ripe old age. I can myself, at any rate, supply one case of a lady who had acute rheumatism at the age of 17, and her mitral valve became then affected, and now, at the age of 75, she still has a loud mitral systolic bruit (to which I have myself listened), and in the interval she has lived an active life, and has borne eight healthy and still living children.

The second direction in which we may be brought in contact with this question of duration of life is where we are asked by friends and relatives, "Doctor, if he gets over this attack will he ever be able to get about and live with a heart so diseased?" Many circumstances will modify our answer: his occupation, his home, and its domestic atmosphere; in fact his environments generally. The whole subject is very full of difficulty, and the question cannot be answered off-hand. Absence of physical labour is certainly not the only thing to be thought of, for it is a fact of which I have ample proof, that women die directly of heart disease at an average age less by some four or five years than men. I shall allude to this again in my remarks on treatment. Sir Andrew Clark says, in the "British Medical Journal," that we must largely modify our opinions as to the gravity of valvular lesions when compensation has occurred; his observations were called forth by the large number of cases that came under his treatment in which bruits were present without signs of undue circulatory back pressure. My own statistics, compiled from 956 deaths with valvular lesions present, show that 47.43 per cent. of people thus affected die of some purely independent affection, such, for instance, as cancer, accidents, etc.

Let us turn now to the second aspect of prognosis: What features does a case of acute valvular trouble present as points from which to judge of the probability of immediate recovery and the supervention of a fairly comfortable existence. These I will take in order, and mention a few details in each.

1. *Cause of the onset of the symptoms.*—This is a point which we are, I think, somewhat liable to overlook; while its importance is perhaps greater than that of any other single factor. In illustration of what I say I will quote two cases in very brief detail. The first was a little girl, æt. 14, seen in consultation in the north of London with Mr. A. A.

Howell. The heart mischief began during an attack of influenza—a disease, by the way, which I believe, often leaves valvular trouble behind it. When convalescent, Mr. Howell sent her to the country for change of air and rest; while there, however, so far from resting, she was given a great deal of hard physical labour to perform, and in consequence returned home worse than she went; when I saw her we agreed that the prognosis was almost hopeless—ascites, general œdema, bloody sputa, with terrible cough, and most distressing orthopnoea, were all present. Under treatment, however, she slowly mended, and eight months later came to see me—grown out of recognition, and though not perhaps the picture of health, still able to thoroughly enjoy life; the heart had hypertrophied, and established, apparently, perfect compensation, though the mitral bruit was still very loud and long. The second case had a different ending: it was that of a young labouring man who came to me as an out-patient at the London Hospital. He had given up his situation some six weeks previously for reasons totally unconnected with his health, and it was during the time when he was doing nothing at home that his symptoms developed. When I saw him his complaints and appearance were not nearly so bad as in the first case, but as they seemed to be due rather to inward tissue failure than to outward extra stress I felt compelled to give a cautious prognosis. He died less than three weeks later. These two cases justify a vague general rule, "If we can find a definite cause for the onset of acute symptoms of cardiac failure we are justly entitled to hope that the case will improve, provided that cause can be to a large extent removed; but if the symptoms appear without any obvious cause, such as bodily exertion or mental trouble and worry—the latter, perhaps, even more important than the former—it would be well to be cautious in what we say about recovery till we have watched the case for a few days."

2. *Results of treatment.*—To discuss this as a help to prognosis seems at first like begging the question, and one might say, of course, if he gets well, the prognosis is good. That is true, but I mean to refer to the primary effect—to the result of the first few days of treatment. Does the pulse become fuller and firmer? Does the heart soon become more regular and slower in its action, and seem to empty itself better? The answers to these questions will probably give us some indication as

to the reserve of strength left in our patient and his cardiac muscle. If signs of response to a lessened strain and to the influence of drugs soon show themselves, we may certainly hope that a new balance of compensation will be struck. If, on the other hand, there is a delay in reaction, then every week, or day even, of this delay will make us more anxious about the ultimate issue of the struggle.

3. *Heredity*.—Dr. Broadbent believes this to be a factor of considerable importance in prognosis, but I cannot help thinking that mental influences are here busily at work, and that it is the knowledge of a mother's or father's death of heart disease which is of greater importance than the fact itself—fear and apprehension are great obstacles to us in our battle with death. One must admit readily enough that a rheumatic inheritance is a factor in predisposition to endocarditis, but that is a different matter to the influence of that endocarditis on the prognosis of an individual case.

4. *Albuminuria*.—From a consideration of the details of over 900 fatal cases, I obtained somewhat contradictory evidence on this point. In the first place, I find that about one-third of them do not show a trace of albumen even up to the end, *per contra*, I find that about one-third of all cases of old standing valvular disease die of kidney mischief, large white kidney is the most frequent form, acute nephritis is also very common as a cause of death, and distinct cirrhotic kidney, with its attendant complications. I was struck, too, by the large number of cases which presented signs of long passed embolic changes in the kidney. It is probable that we may thus sum the matter up: if albumen be found in the urine it indicates undue stress on the kidney, and if blood be present it points to embolic affection of that organ; but neither the one nor the other *alone* need cause us undue alarm, and is not necessarily of gloomy significance, but if, on the other hand, casts are present to any great extent, indicating as they do a disintegration of renal epithelium, they must be looked upon with very grave significance as an individual factor in the case, apart from other conditions.

5. *Emboli*.—Cerebral softening of a fatal character due to emboli I find was present in 40 cases out of 950, but there was evidence of old softening in the same proportion, so that the danger from this source while considerable is not overwhelming. As regards emboli of organs other than the brain, I have no evidence that they are, *per se*, fatal, but there is strong evidence that those cases in which

emboli were found, offer a younger average age by full five years than those in which they were not present. This is a fact which we cannot ignore, and with a history of infarcts we must look with a graver eye on the future of the case, though the immediate prognosis need not be necessarily gloomy.

6. *Principal features of the case*.—Amongst the more definite symptoms which seem to be grave in their significance, and which occur most frequently amongst my statistics of 450 male deaths, are, (a) sudden unexpected death, 48 cases. I mention this that I may state that in three of these the aortic valves were perfectly healthy. (b) Severe pulmonary hæmorrhage, 23 cases. (c) Anginal attacks of pain, 15 cases. (d) Severe vomiting and diarrhoea, unchecked by treatment, 43 cases. (e) Dyspnoea, curiously paroxysmal in character, gangrene, and severe purpura also occur; but (f) the largest group, numbering 73 cases, presented features of simple cardiac failure, without evidence to show that one organ felt the failure in circulation more than another.

7. *Bruits present*.—Except for the possibility of sudden death in aortic disease (and even then the pulse will tell us as much as the stethoscope), against which possibility we may warn the friends, the less said about the bruits in connection with prognosis the better.

Prognosis then is a very difficult matter; and even after carefully weighing all the above factors, we shall still, I do not doubt, make many mistakes.

On the subject of treatment I speak with some diffidence. I have no new specific for a heart worn out by valvular disabilities, no new cardiac tonic to offer to your criticism. I only propose to bring forward for discussion a few scattered remarks on old drugs and methods of treatment, laying stress on some points which experience has taught me are of most importance.

The first observation that occurs to me is to protest against a too early exhibition of Digitalis or other direct cardiac drugs. We must remember that the anatomical defects, against the results of which we are fighting, are irremovable; if Digitalis be our last and highest hope against those results why bring it to the front too early? Surely it is more rational to commence our lifelong fight against cardiac failure with the simplest remedies we possess: with bodily rest, with the evacuation of a loaded colon, with a stimulant to the appetite, by so doing we not only spare the heart from the

possible—some would say from the probable—deleterious influences of *Digitalis*, etc., but we gain a very valuable practical hint as to the recuperative powers of its musculature, by noticing the progress of symptoms when difficulties are removed from the circulatory path. On these grounds, when acute symptoms of failure are present I prefer to rest the damaged organ, and to watch results for a day or two under the influence of a few doses of Soda and Gentian with Magnesium Sulphate.

This plan is by no means opposed to Oertel's method of treatment by graduated exercise, for the latter refers not to acute cases, but to those in which compensation is moderately good, or, at any rate, improving, and the patients are able to get about with tolerable comfort. In support of Oertel's views, I may call to your minds a statement I made in connection with prognosis, viz., that women die, on an average, five years earlier than men under the same affection of valvular disease. This might be due to several causes, to the greater emotionability of women making greater taxes on the cardiac power, to the earlier age of women in respect of their liability to rheumatism, etc., but to the latter I would reply that the evidence of their earlier liability is by no means conclusive, and to the former that my statistics are taken from a class of women amongst whom the emotions are not cultivated. Till a better explanation offers itself I conclude that it is in some way concerned in the difference of occupation, and the fact itself is a great testimony to the truth of Oertel's views on treatment.

Diet. Why should we forbid a cardiac patient to eat or drink what they please? The appetite, while acute symptoms are present, will, I feel sure, prevent them taking an injurious meal of solid food, and for the rest the patient's own previous experience of his stomach is better than any theories of ours. As regards quantity of fluid, Foster distinctly tells us that even by direct intravenous injection we cannot raise blood pressure except by foolish lengths of experiment, much less then are we likely to do so through the mouth; for absorption from the alimentary canal into the blood is much more under the control of cell vital activity or cell instinct than of any physical laws of osmosis. That the absolute total quantity of blood in the body—governed in its distribution as it is by vasomotor regulations—can within ordinary natural limits make any difference to the heart

I fail utterly to believe. Hence, neither from reasons connected with pressure, nor with quantity can I see any reason for depriving a patient of that amount of liquid which his wishes dictate, provided its bulk in dose at one time is not great enough to mechanically interfere with the stomach. For compensation to occur, good nourishment is required, and if driven to extreme I would rather fatten than starve my patient.

Sponging with tepid or hot water. I am often tempted to think that those who rush to Antipyrin, Sulfonal, Phenacetin, etc., for the reduction of temperature, or the production of sleep have too little experience of the antipyretic or sedative effects of tepid sponging of the face, chest, and neck, especially the carotid regions of the latter. I have often found several hours refreshing sleep given by these simple means; nor must we forget the natural anxiety of a mother, or a wife, to be actively engaged in doing something for the relief of a beloved object, a wish which is admirably met by this simple expedient.

Bleeding. Do we bleed often enough in acute cardiac cases? I am inclined to think that the operation has gone too much out of practice. In the case of the little girl I mentioned earlier, three or four leeches applied to the præcordium, with subsequent encouragement of the bleeding by warmth, exerted a marvellous effect, the cyanosis and orthopnoea rapidly yielded, and I firmly believe that the hæmorrhage saved her life. These two features of cyanosis and orthopnoea are the chief indications for the abstraction of blood; the operation is not done to permanently reduce blood pressure, which it cannot do, but to temporarily relieve the right heart, a purpose it admirably fulfils.

Drugs. Digitalis. With regard to this old friend, I merely draw renewed attention to the use of the powdered leaves or infusion when the urinary secretion is especially scanty, under which condition they are far preferable to the tincture. I would like too, to ask for any actual experience of its so-called cumulative action, my own belief is that no such action is to be recognized unless it be given improperly. If the urinary secretion be free, I hold that the cumulative action will not be witnessed; if it be not free, and *Digitalis* be given to assist, then I hold that the drug is being given improperly if it be pushed after the first six or eight doses have been found to be of no avail, and the drug must then accumulate in the blood precisely as would any other which is non-volatile,

and is being poured in at one end with no channel of elimination at the other.

Of *Strophanthus* I have had considerable experience amongst out-patients; the great caution is not to give it too freely; three to five minims of the tincture I have always found sufficient, larger doses being apt to produce great cardiac distress. It certainly has not so much tendency to upset the stomach as has *Digitalis*, and is said not to act so much on the arterioles; it is certainly inferior to *Digitalis* in diuretic action.

Nitrite of Sodium. Dr. Brunton tells us that this drug dilates arterioles, and so diminishes the heart's work. I have used it very frequently in the last year or eighteen months, taking as the chief indication for its exhibition any form of chest pain associated with evident cardiac failure. I have been very pleased indeed with the results, and I know several patients who have obtained so much relief from it that they do not like to give it up at all. I believe the drug to be worthy of much more extended use; it is also cheap to buy, and stable in its composition.

Opium. This drug is often said to be contra-indicated in valvular heart disease, but I strongly suspect that its depressant action is like the cumulative action of *Digitalis*, and only appears on the abuse of the material. In my experience Opium is simply invaluable in heart disease; it calms the restlessness, it removes that intolerable sense of oppression from which so many patients suffer, it promotes sleep; let us not forget that restlessness and sleeplessness are symptoms of bad omen; try by all simpler means already mentioned to combat these symptoms, but if they fail, give Opium without hesitation, say, a grain or a grain and a half, before bedtime, for one or two nights: the sleep procured is a better tonic than any drug. I say, without fear of contradiction, Opium given with deliberation and watched with intelligence, is perfectly safe.

A final word against astringents in hæmorrhage. Could they be applied locally to the bleeding vessel, all well and good, but acting through the mass of the blood they can only—if they have any action at all in this way—increase blood pressure generally; then, ask yourselves, which vessel will be likely to feel this pressure most injuriously—surely the diseased one which has already ruptured.

I have now very briefly sketched the diagnosis, prognosis, and treatment of valvular disease of the heart. I have, I trust, brought several points to

your notice on which discussion and criticism may be exercised. Several opinions I have expressed border, perhaps, on the unorthodox, but I hold that we cannot get truth without boldly questioning for ourselves traditional teaching; and if to-day I have encouraged some of you to independent thought on heart disease my intention has been amply fulfilled.

ON TOO FREQUENT MICTURITION IN WOMEN.

A Lecture delivered at the London Hospital, by
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Obstetric Physician to the Hospital.

THIS symptom is often denoted by the term "irritable bladder." The words should rather be, as Dr. Matthews Duncan pointed out, "irritated bladder," because every bladder is irritable, that is, capable of being irritated.

Too frequent micturition must be distinguished from incontinence of urine. In both conditions the patient may say that she cannot hold her water, or that the water is constantly running. But in incontinence the urine is continually flowing away as fast as it is poured into the bladder; while in irritated bladder the urine is retained for a little while, but the call to void it comes too often.

Most of the conditions which cause painful micturition also cause it to be too frequent. This is the case with all inflammatory diseases of the pelvic organs. But not all causes of frequent micturition produce pain in micturition. I have in a former lecture put before you the causes which produce painful micturition; in this one I shall not further refer to them, but speak only of those which cause micturition to be too frequent, but do not make it painful.

Some of the conditions which produce too frequent micturition are associated with physical signs of disease of the pelvic organs; others are not. Some of these conditions are associated with changes in the urine; others not. In some of them the irritation of the bladder is only occasional, the patient sometimes having to pass water with annoying frequency, sometimes being able to go as long as she wants to: in others the irritation is continuous. In some of these latter the trouble is of recent origin, in others it dates as far back as the patient can remember.

Cases in which frequent micturition is the main symptom may be divided, for convenience of clinical study, into four classes :

1. Those in which there is *no local disease* appreciable by the ordinary methods of local examination.
2. Those in which vaginal examination reveals morbid changes in *the uterus or its appendages*.
3. Those in which no disease can be detected without examination of *the urethra*.
4. Those in which the cause cannot be made out without examination of *the urine*: that is, those dependent on disease of the bladder or kidneys.

Consider, first, the cases in which you find *no local disease*, neither of the uterus, nor the urethra, nor any morbid state of the urine.

Micturition is generally more frequent in *pregnancy* than at other times.

In certain *nervous diseases*, such as locomotor ataxy, the bladder is more irritable than in health. In this disease, although the urine is healthy and the bladder not painful nor tender, yet the call to micturition is produced by less tension of the bladder than in health. Duchastelet (*Annales Med.-Chir.*, Paris, 1888, p. 112) has shown by experiment that while in health the call to micturate occurred with a pressure of urine in the bladder represented by + 30, in ataxy a distension represented by + 20 put the patient in pain. The condition is a hyperæsthesia of the bladder.

There are some women who are all their lives troubled by being unable to hold their water so long as they would like to. The irritability is not enough to be a serious source of annoyance to them, except so far as this, that they cannot go into society, on excursions, to entertainments, etc., unless the arrangements are such that they can frequently retire. They may date this back as long as they can remember, and be unable to assign any cause for it. In such cases, the condition may be, as Dr. Matthews Duncan suggested, that the *bladder* is congenitally *small*, and will not hold quite so much urine as that of most women. Duchastelet (*"Annales Med. Chir.,"* Paris, 1888, p. 117) has brought forward some evidence to show that the cause of frequent micturition really is smallness of the bladder. In a patient who micturated twice daily, 850 grammes of fluid, with a tension represented by +85 could be got into the bladder before pain was produced. In another patient who micturated five or six times daily, 400 grammes, with a tension of +30, produced pain. In these cases there are no physical signs of disease,

there is no pain in micturition, nor alteration in the urine. The irritability of bladder is apt to be made worse by various temporary causes, such as a cold, or a cough, or the drinking of a great deal of fluid.

In other cases in which a similar complaint is made, the frequency of micturition dates from some occasion in which the *bladder* was *over-distended*. Dr. Matthews Duncan relates a case in which punishment for wetting the bed led a child to retain her urine for so long that the bladder became over-distended, and the result was permanent enlargement of the bladder, with excessive irritability of the sphincter, and consequent frequent micturition. I have seen cases in which similar trouble dated from an occasion on which the patient had to go a very long time without emptying the bladder.

Micturition may be frequent, not owing to any local change, or any disorder of the bladder at all, but simply from an *excessive quantity of urine*, so that the bladder gets filled too quickly. Thus, in diabetes mellitus, diabetes insipidus, and in hysteria, there is frequent micturition, simply from quick and frequent filling of the bladder.

There is one kind of vesical irritability different from all the rest, viz., *nocturnal enuresis*. This means that the patient at night, while asleep, involuntarily and without knowing it, passes water once or oftener during the night.

This disease appears to be due to irritability of the spinal centre presiding over micturition. It is a nervous disease; not one depending on any disease of the urinary apparatus. It often runs in families—several members of the same family being affected with it. It is sometimes inherited, and a tendency to other nervous diseases, such as migraine, epilepsy, insanity, hysteria, etc., will often be found to run in the family. It occurs chiefly in children, and usually gets well or is cured before puberty; but sometimes it lasts until after puberty. The ejaculation of the urine does not depend on the quantity of urine in the bladder, but on the state of the nervous centre. It resembles nocturnal seminal emission in the male in being a spasmodic affection, due to weakness of the spinal nervous centre. The centre which presides over micturition is situated about opposite the sixth dorsal vertebra. Nocturnal enuresis may be produced by reflex irritation, such as vulvitis (or in the male, phimosis), by worms, fissure of rectum, or piles (although these latter are not common in young females), ulcers, calculi, or growths in the bladder, or by too acid urine.

In the treatment of this disease, such causes of reflex irritation must be sought for and removed. Over-exertion, by weakening the spinal centre, may favour its occurrence; therefore this should be forbidden. Having removed any exciting cause, try to break the morbid habit, by directing a nurse to wake up the patient, and make her get up and empty the bladder at regular intervals during the night. The younger the patient, the more successful this treatment will be. If it is not quickly successful, aid it by the following measures:—

Let the patient sleep on a mattress, as lightly covered as the weather will permit. Forbid a large drink before going to bed. Give Belladonna—as much as two grains of the extract may be given for a dose. Order the cold douche to lower part of spine. If Belladonna fails, prescribe Chloral at night as a suppository, administering about twice the dose in this form that you would give by the mouth (the dose must be regulated by the patient's age). Give also general tonics, such as Cod Liver Oil, Arsenic, and Steel. The Bromides are useless.

In any case of irritated bladder, in which the history does not supply a sufficient explanation, or in which a definite opinion as to the presence or absence of local disease is sought for, you must make a complete examination. Look at the parts. Make a vaginal examination. Pass a catheter. Examine the urine.

Never forget that very frequent micturition, or even complete incontinence of urine, may be a symptom which indicates *retention of urine*. Hence when you are told that the patient has within the last few days been unable to hold her water, never omit to *pass a catheter*. The passage of a catheter is not only indicated to relieve retention, which may be present, but also to ascertain the condition of the urine. Urine passed naturally by a patient may be mixed with blood or pus derived from the vagina. Draw the urine off with a catheter, and you get it as it is in the bladder.

Imagine now a case of too frequent micturition in which you find nothing wrong with the urethra, and the urine is healthy. Irritation of the bladder may be due to slight *local causes*. Thus I have known a tampon of cotton wool allowed to remain in the vagina irritate the bladder so that micturition was too frequent: the proof being that when the wool plug was withdrawn the patient could go as long as usual without micturating. Matthews Duncan

mentions the case of a urethral cyst which did not affect frequency of micturition until it was cauterized, and then micturition became frequent until the effects of the caustic had passed away. An ill-fitting vaginal pessary may make micturition frequent. I have known the bladder to be so irritated by the stitches after an operation for vesico-vaginal fistula, the result of which was complete success, that during the first few days urine was passed so often as to make the nurses doubt whether the fistula had been closed. Hæmorrhoids may cause too frequent micturition.

If there be inflammatory disease in the pelvis, or if there be a displacement of the uterus, that is sufficient explanation of frequent micturition.

There is a kind of bladder irritation met with chiefly in women who have had children. This results from relaxation of the structures forming the pelvic floor. Some women who are at most times free from local discomfort, when they get depressed in health from any cause, as, for instance, from a cold, or from several disturbed nights, get backache and irritation of the bladder. I think this is due to a slight temporary prolapse of the pelvic floor. The degree of prolapse is so slight that it is not enough in a woman with a healthy nervous system, to irritate the bladder; it is not enough to be detected by examination, because it is not more than is often seen in healthy women. There must be a morbid change somewhere to account for this temporary irritability of bladder; and a temporary yielding of the muscles forming the floor of the abdominal cavity is the most reasonable explanation. It is a condition analogous to the temporary aching and watering of the eyes which troubles hypermetropic patients when they get out of health, and which is due to temporary weakness of the ciliary muscle. This kind of irritation of the bladder causes a constant dribbling of small quantities of urine during the day, not a spasmodic ejaculation at night. There is no strong stream. The patient is not disturbed by it while recumbent. Escape of urine is often promoted in such patients by coughing. This kind of bladder trouble is benefited by Strychnine and Ergot.

There are other cases in which the same change, viz., slight prolapse, is more marked, and then the troubles which it causes are present continually, not only for a few days at a time. The patient tells you that ever since some more or less definite date (usually that of a confinement) the bladder has

been more irritable: that greater frequency of micturition has been at all times present, though sometimes worse than others. With this there is generally some aching in the back and lower abdomen. Most are aware that well-marked prolapse causes these symptoms; but remember that these symptoms may be present, and may be due to a slight degree of prolapse, without the patient being in the least aware that anything comes down. The precise changes in the position of the pelvic viscera which the prolapse causes are immaterial: whether the pelvic floor bulges down without relative descent of the uterus, whether the uterus descends without other change of shape or position, or whether it be unduly anteverted or be retroverted (unless in the latter case it be congested also) makes no difference.

The diagnosis in this class of cases and in the one spoken of before, is made by the character of the symptoms, together with the physical signs.

The characteristic features are these:—1. The absence of pain on micturition. 2. The complete relief to the bladder irritation when the patient lies down. 3. The absence of all other symptoms beyond the backache, etc., common to all forms of prolapse. The physical signs are those of the change in the parts forming the pelvic floor, with absence of any signs of disease of the urethra, and of any abnormal constituent of the urine. If the symptoms are temporary only, it may be expected that by restoring the patient's nervous tone, these symptoms will be relieved. If they are continuous, and examination shows a slight degree of descent, slight cystocele, or anteversion, or retroversion, then the support of a pessary may be advisable. Hewitt's cradle pessary is especially useful in these cases.

Dislocation of the urethra: its being pulled upwards either by the gravid uterus or by tumours, or dragged downwards by cystocele or prolapse of uterus are described by Baker (American System of Gyn.), the latter as a cause of irritable bladder. The gravid uterus does not pull up the urethra until labour has begun. Such pulling up of the urethra as occurs with tumours, unless accompanied by pressure on the urethra or by much congestion of the pelvic organs, does not irritate the bladder or cause any trouble. Prolapse of the urethra occurs with cystocele: but apart from cystocele I have never seen it. I am not aware of any difference in the symptoms of cystocele which depends upon the greater or less extent to which the urethra is

dragged down with it. According to Baker, "settling of the uterus, or of an ovarian cyst after puncture" may cause the lengthened urethra to double upon itself so that a sharp angle is formed in its course. This I have never seen.

By looking at the parts you will discover the presence or absence of disease of the meatus, or of the vulva, or of vaginitis or vulvitis.

Frequency of micturition may be due, in the female as in the male, to *stricture of the urethra*. You will ascertain this by passing a catheter, or rather by attempting to pass one. The female urethra is bigger than the male: No. 14 male catheter will usually pass the female urethra quite easily. A female urethra which will not let No. 10 male catheter pass is the subject of stricture.

Stricture of the urethra is less common in the female than in the male. It is important to identify it when it does exist, because the result of treating it is satisfactory.

There may be congenital smallness of the meatus. I have not seen a well-marked example of this; nor have I been able, in my own experience, to connect smallness of the meatus with frequent micturition. Such congenital smallness is not dependent on disease, and, therefore, I do not suppose interferes with function. Horrocks (Obst. Trans., vol. xxix., p. 50) mentions a case in which it was impossible to pass a catheter of any sort, and in the patient, a girl aged 19, there had been "difficulty of micturition, chiefly incontinence, from birth," and there were "obvious congenital deficiencies;" but it is not stated what these were. Atresia of the urethra is sometimes met with as a congenital condition in infants; but it is obviously incompatible with life unless the urine escape by some other channel—usually a pervious urachus. These great malformations are quite different from mere smallness of the urethral orifice in an otherwise well-developed patient.

The most common cause of stricture in the young and middle-aged of the female, as of the male urethra, is gonorrhoea. It is rarer than in the male; but such numerous cases are on record* that the fact cannot be doubted.

Another kind of stricture arises from injury to the urethra during difficult labour, leading to sloughing and the formation of a cicatrix, narrowing the urethra. These are rare, because the sloughing which follows labour more often leaves a vesico-vaginal fistula.

* See Author's paper "Obst. Trans." vol. xxix.

The urethra is sometimes found converted into a thick fibrous cord, and its lumen much narrowed. I have suggested that as the urethro-vaginal septum is the homologue of the prostate gland in the male, this form of stricture, which occurs in old women, may properly be regarded as analogous to enlargement of the prostate in the male. I have known the urethra so narrowed by stricture of this kind as to grip a catheter so tightly that, when its withdrawal was attempted, the catheter, a gum elastic one, broke, a bit remaining sticking in the urethra.

The cicatrization of a chancre situated on the urethra, may narrow the canal so as to produce the effects of stricture.

Whatever the cause of stricture, irritation of the bladder and excessively frequent micturition is the consequence. The diagnosis is made without difficulty by passing a catheter. An instrument of the ordinary size will not enter. To get into the bladder, you have to use a small instrument, and this is tightly gripped. In passing the catheter, do so slowly. Note carefully the amount of pain produced. There may be hyperæmia of the urethra and neck of bladder. The severe pain caused by catheterization will point to this.

The treatment of stricture of the female urethra is simple. It is to dilate the stricture with bougies. The canal is so short, and so easily felt through the vagina, that there is no difficulty in doing this. Irritation of the bladder, due to stricture of the urethra, is cured by dilating the stricture.

In all cases of frequent micturition in which the symptom is too troublesome for a rough diagnosis made from the history to be sufficient, pass a catheter, draw off the urine, and examine it carefully. The bladder irritation may be from *cystitis*, or it may be from *disease of the kidney*. Frequent micturition is a symptom of Bright's disease; therefore if the urine be clear and free from sediment, test for albumin. In pyelitis, and pyonephrosis frequent micturition may be the symptom which induces the patient to seek medical advice. Thus I find among my notes the case of a girl, æt. 18, who went to a general physician complaining of pain in the lower abdomen, and frequent micturition. These being her complaints, he judged it probable that there was disease within the pelvis, and sent her to me. I found nothing abnormal within the pelvis, but the urine loaded with pus, and great enlargement of the right kidney.

A surgical colleague was consulted, the kidney removed, and the patient cured.

The presence of suppuration at some part of the urinary tract is sufficiently indicated by the presence of pus in the urine. The source of the pus is indicated by the facts that in cystitis the urine is generally ammoniacal and alkaline in reaction. In pyelitis, the urine is usually acid; so that if the urine contains much pus, but is acid in reaction, this is a point in favour of pyelitis. But the cystitis may be slight or very old; and in that case the urine may contain pus and be acid. In acute pyelitis there may be in the urine epithelial cells of transitional character: and this, if the cells are numerous and well characterized, will be proof that the pus comes from the kidney. But in chronic pyelitis these cells may not be present: so that failure to discover them is no proof that the kidneys are healthy. In pyelitis there are more direct signs of irritation in the loins: pain in the loin on the affected side; aching and weakness in the back—these symptoms being increased by pressure. In the ordinary backache, which is so common in women, pressure relieves. There may be a history of renal colic. If there be a tumour in the situation of the kidney, with pus in the urine, the source of the pus will be clear. In pyelitis there are often rigors and hectic fever; in cystitis much fever is not common. A conclusive proof that the pus comes from the kidney is the observing that the pus is sometimes abundant, sometimes slight in quantity or absent; and that with these variations there goes alteration in the size of the renal tumour—a diminution in the tumour following a copious discharge of pus.

When the amount of pus is great, the urine acid, fever and wasting marked, and the loins tender on pressure, the probability is that the pus comes from the kidney. When there is a tumour the diagnosis is not difficult.

We may get both cystitis and pyelitis, or cystitis and Bright's disease. In such cases the diagnosis, as to how far the symptoms are due to the bladder and the renal trouble respectively, will be very difficult.

If either of these conditions be present, the bladder irritation is a symptom quite of subordinate importance. It is necessary to mention them here because the great frequency of micturition may be the symptom which leads the patient to seek advice. The treatment of bladder and renal disease is too large a question to be entered upon here.

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A LECTURE ON DROPSY.

Delivered at Guy's Hospital, December 2nd, 1893, by
P. H. PYE-SMITH, M.D., F.R.S.

TO-DAY, Gentlemen, I propose to speak from what may be called the analytical point of view (the point of view from which we lately considered hæmorrhage and dyspnœa), of the very common and important symptom of dropsy. We shall not start with its causes, but with a case of dropsy, and try to see how to manage, treat, and cure it.

A word to begin with as to the physiology of dropsy. The word itself is a mere vernacular corruption of *hydropsia*, the "water-disease." It was known as such to the Greeks, and has been known ever since by that name. The Germans call it *wassersucht*, "water-disease"; and the English terms are "water on the brain"—*hydrocephalus*; "water on the chest"—*hydrothorax*, *hydropericardium*; and water in the abdomen—for which we generally use the Greek word *ascites*, the belly being filled with water like a wine-skin. When the effusion was all over the body the Greeks called the effusion *anasarca*, *i.e.*, "water over the whole flesh," general subcutaneous dropsy.

What do we mean by dropsy? we mean effusion of water; but then there are two kinds of effusion of water. If you injure your finger you will find that all round the hurt water begins to gather; if you have a boil which is suppurating, you will find a considerable ring of oedematous tissue round it; that is exudation of water. If you prick the swelling you will find there is a clear watery fluid in it filling the tissue. That is not blood; it consists only of certain parts of the blood which have exuded, these being the water, the albumen and globulin, the salts, the pigment, and minute quantities of extractives, as they are called, urea and other crystalloids, but not the fibrinogen, nor the leucocytes and red corpuscles. Now this is a different kind of effusion from what we call dropsy. Dropsy is passive effusion, the other is inflammatory: or, as is sometimes said, the one is acute, active oedema; the other is chronic, passive oedema. We speak of the latter only as dropsy;

but it is very difficult to draw a sharp line between the two; they run into one another. A man has water on the chest; and now and then you find *post mortem* pure serum—no clots and no pus, the surface of the pleura being quite free from false membrane: it is a pure dropsy. But much more frequently you find evidence of inflammation—"false membranes," and more or less turbid serum, perhaps tinged with blood. And there is every gradation between inflammatory exudation and passive dropsy. We will not, then, attempt to draw a line where Nature has not drawn one, but admit that the two often co-exist, passive exudation from pressure on the veins with active exudation from inflammation.

What is the immediate mechanism of dropsy? You see a man with the whole arm swollen. You immediately look to the upper part of the arm to find if something has not been pressing on his axillary vein; and you generally find that a tumour, or the handle of a crutch, or possibly a splint, or ligature, has been mechanically squeezing the great vein that returns the blood from the arm; and the result of that pressure is to produce the dropsy. Now try that in the laboratory. Take a rabbit's leg and put a ligature round the femoral vein. You do not produce dropsy; because the lymphatics take up the fluid as fast as it is exuded from the capillaries into the lymph spaces. If you tie the lymphatic trunks also, then you get dropsy. Again, when there is oedema in one limb of an animal you find that by moving the limb about you can prevent or remove it. The movement expedites the flow of lymph, and the removal of the lymph makes up for the exudation. Dividing or not dividing the principal nerve also makes a difference, so that probably there is some influence of the nervous system which governs the exudation and the absorption of lymph.

We can understand, then, that dropsy depends upon pressure on the returning vein. Apart from that, it is difficult to explain dropsy. It does not depend upon osmosis. Osmosis is the phenomenon, investigated by Graham, in which two fluids put on opposite sides of a membrane will gradually diffuse through with varying degrees of rapidity. At the one end of the scale you have dilute Hydrochloric Acid, followed by a solution

of common salt, then various other crystalline solutions; at the other a solution of albumen, gum, or other colloids: the first diffuses readily; the last very slowly. Even peptones, though they diffuse rapidly compared with albumen, diffuse very little compared with saline solution. That is the law of osmosis; but it has nothing to do with pressure. Diffusion of fluids under pressure is what takes place in the human body; but it is not merely a question of pressure. The investigations of Professor Heidenhain, of Breslau, have thrown remarkable light upon this subject lately, showing that a great deal of what we know as exudation and absorption depends, not upon purely mechanical principles, but on the active work which is probably done by the endothelial plates of the blood-vessels, and, possibly, by those which line the lymphatic spaces; for it seems probable that the protoplasm of these plates retains some active properties. That opens a very wide field. Dr. Starling has been working in Heidenhain's laboratory, and I have no doubt we shall learn more on the subject from him. Our late friend, Dr. Wooldridge, once said to me, "You know nothing at all about dropsy." I told him I should be glad indeed of further instruction; and, I have no doubt that, with his genius and experimental skill, he would have told us a great deal about it.

Remember then that the pathology of dropsy is a difficult subject. Still it remains that in a large number of dropsies we can recognize a mechanical cause—pressure on the returning veins, and that in many we have a certain amount of inflammation along with passive dropsy.

Without further preface we will go on to the diagnosis and treatment of the different kinds of dropsy.

1. Local dropsy. If one arm is swollen that will almost always depend upon pressure on the axillary vein. If both legs are swollen, and there is no evidence of dropsy in any other part, that will almost always depend upon pressure on the vena cava, with great thrombi forming in the two iliac veins. Every now and then you will see such a case—rare and curious, but not difficult to decipher if you remember your anatomy. Sometimes, again, the whole of one side of the face and neck and of one arm is swollen. I had such a case in Philip Ward a year ago. The man came in with that condition, but without any other important symptoms. No doubt there was obstruction

of one or more of the great veins, perhaps the right innominate. The veins on the front of his chest were enlarged and tortuous. The external mammary and the superficial epigastric veins instead of being, as in a healthy person, things you could not see, were great varicose vessels as big as the internal saphenous vein before it enters the femoral. Those are rare cases of local dropsy. With regard to their treatment, I would say that almost always they depend upon local pressure; and almost always you will cure them if you can relieve that pressure. In the case of one arm or leg you can do so; when both legs or the head and neck are affected, it depends on pressure on deeper veins, and you cannot do much. Sometimes you may attempt their relief by puncture, mechanically letting free the effused serum. In all cases remember that gravity is always acting. When you have an œdematous hand, if you hold it up the tension ceases. Whenever you can get gravity to act use it. The consequent treatment by elevation is particularly useful in what is called elephantiasis or Barbadoes leg (case in Mary Ward).

2. Portal dropsy is also a local dropsy; but it is local over a very wide area. It follows pressure on the portal vein. In most cases the pressure is produced by cirrhosis of the liver (interstitial hepatitis) contracting and squeezing the portal capillaries; but anything, whether hydatid, cancer, enlarged gland or adhesions, which squeezes the trunk of the portal vein or its distribution in the liver, will have the effect of raising the pressure in its capillaries, and producing this portal or hepatic dropsy. You know it at first sight because it is a dropsy below the diaphragm; all the parts above the diaphragm are free, all the parts below are affected. It first affects the abdomen, producing ascites, and, secondly, the legs by pressure of the effused fluid upon the inferior cava. Now and then one may be deceived by the history. I remember a patient with ascites and swelling of the legs, who assured me her legs were swollen before her abdomen. It was puzzling, and one only got a clear diagnosis by disbelieving her. People notice the condition of their legs much more quickly than that of their abdomen, especially women; for what with pregnancies, obesity, and flatulence they are accustomed to the belly becoming larger, and do not notice it, but swelling of the feet, by making their shoes tight, forces itself on their notice. As a matter of fact, the ascites

always comes first, and the dropsy of the legs follows; then you have the complete picture of portal dropsy:—a great swelling of the abdomen and swelling of the legs, the genitals being unaffected, and no dropsy above the diaphragm.

Why should we have the effusion into the peritoneum? Because, you say, there is increased pressure in the capillaries of the portal vein, which are distributed to the intestines. True enough; but why is there not hæmorrhage into the abdomen from the same cause, since you get hæmorrhage into the stomach from cirrhosis, and great swelling and hyperæmia of the spleen. Why should the same cause—constriction of the portal trunk and high pressure in its branches—produce hæmorrhage into the stomach, and exudation of serum into the great cavity of the peritoneum? I cannot answer the question; but it requires answer, and we ought to think over it. To my mind dropsy is an affair of the lymphatics, and wherever you have endothelial plates there you may have dropsy. Dropsy of the pleura, dropsy of the pericardium, dropsy of the peritoneum, dropsy of the tunica vaginalis; these are all parts of the great pleuro-peritoneal cavity lined with epithelial plates from the mesoblast, all similar in constitution, all with stomata, all in direct connection with the lymphatic system. They are great lymph sacs like the lymph spaces under the frog's skin. These are the chosen seats of dropsy. Now, where else do we find dropsy? Under the skin, and to some extent under mucous membrane. But what is the subcutaneous and submucous tissue? It, again, is lymphatic tissue, full of endothelial plates and stomata, with abundant lymphatics connected with it, and more or less accumulation of adenoid tissue here and there. You may compare dropsies in the serous cavities and in the skin with the absence of dropsies in the solid organs. The liver, kidneys, suprarenal capsules, brain, thyroid, heart, muscles, bones, cartilages—all these are strikingly exempt from dropsy. You will say there is one viscus which often becomes œdematous, namely, the lung. Quite true: but then, surely, the lung is just the part that is full of connective tissue with lymph spaces.

How do we deal with this portal or hepatic kind of dropsy? We must interfere for the relief of the patient; for portal dropsy, if not relieved, will prove fatal. If you can relieve it, and if the cause is not irremediable, if, for instance, in a case of cirrhosis, the patient will give up drinking, I

believe you may give a tolerably good prognosis, though a guarded one.

I have seen several cases tapped more than once; and—what has struck me very much—I have seen cases of cirrhosis which have been only accidentally discovered; so that I would encourage you to treat these cases. A certain proportion you will cure, supposing the patient gives up his intemperate habits. The first thing is to get rid of the effused serum mechanically by tapping, or, what is pleasanter, by carrying it off through the great emunctories, through the kidneys, the skin, and bowels. The lungs ought to help us, for they get rid of a great quantity of water as vapour; but we cannot increase that amount. On the other hand the bowels which in health only remove a very small proportion of the water from the body can be made by actively purging to remove a great deal. So that we depend first on tapping, secondly, on diuretics, diaphoretics, and purgatives. In hepatic dropsy as soon as there is enough ascites to make you sure of not wounding the intestines it is time to tap. Drugs will act very much better afterwards. The best are undoubtedly the diuretics. You may use any kind you prefer; only take care to measure the amount of water passed, and see that you get double or treble the amount previously excreted. You may use the Acetate of Potash, Squill, and Broom which make our diuretic mixture; or you may give Resin of Copaiba, in reference to the use of which Dr. Taylor published some remarkable cases in our Reports. More stimulating diuretics, such as Sweet Spirits of Nitre, Digitalis, and Squill, may be administered, or you may prescribe a very excellent diuretic—water in large draughts. A pleasant diuretic is what we call imperial drink, *i.e.*, lemonade made with Cream of Tartar. I remember the case of a navvy who came into John Ward when I was clinical clerk there. He had great ascites, but was otherwise strong and healthy, and he absolutely refused to be tapped. So Dr. Gull gave him this imperial drink. It was very warm weather, and as the patient could not get any stronger liquor he drank it as if it had been beer. He got rid not only of all the lemonade which he drank but also of the serum in his belly. In less than ten days all the fluid had disappeared, and he went out in triumph at having escaped the tapping.

These are the methods, then, of dealing with portal dropsy—tapping, diuretics, and keeping the bowels open. Do not purge violently, lest it

should cause hæmorrhage from the bowels and other uncomfortable symptoms. A little blue pill at night and saline mixture in the morning is invaluable.

3. Cardiac dropsy is also a mechanical dropsy, from over-pressure in the venous system; only the over-pressure is not produced by a mere local pressure upon a single vein, like the portal. It is due to deficient pressure in the arteries, and increased pressure in the systemic veins over all the body. The over-pressure in the veins and the under-pressure in the arteries depend upon some derangement in the force pump we call the heart. Cardiac dropsy usually depends on valvular disease of the heart, sometimes on secondary dilatation of the right side and tricuspid incompetence, occasionally on adherent pericardium, but that has the same effect. It is not dependent upon fatty disease or functional disorders of the heart.

You know it by its characters. Just as portal dropsy is below the diaphragm, so cardiac dropsy is over the whole body; it is anasarca. The feet swell first, because the patients walk about before they are very ill, and the feet being lowest swell under the influence of gravity. But that does not help us much in diagnosis, since all dropsy affects the feet. Cardiac dropsy, however, affects the arms also, while portal dropsy never gets above the diaphragm. That is an important point of diagnosis. Often the dropsy affects one side only; and that is explained by the way in which the patient lies. If a patient with cardiac dropsy lies all night on the left side, his left hand and arm will be swollen; whereas the opposite will take place if he lies on the right side. The head will be swollen if the patient lies low in bed; but these patients have usually orthopnoea, and prefer to be propped up, so that you seldom see this. In addition to the anasarca, in the later stages of cardiac dropsy you have ascites and often hydrothorax also. You will not see much cardiac dropsy in aortic disease until the later stages, when it becomes clinically mitral.

There is another kind of cardiac dropsy which is very much like this, but different in its pathology. It is secondary. There is no valvular disease; but the right side of the heart dilates in consequence, not of mitral regurgitation, but of pulmonary obstruction, phthisis, emphysema, and bronchitis. There is obstruction to the pulmonary circulation, the right side of the heart dilates, there is over-pressure in the veins, under-pressure in the

arteries, and dropsy follows. That you may call secondary cardiac dropsy.

Another interesting form of dropsy, I believe, is really cardiac, although it is generally called renal. That was explained to my mind, and, as far as I know, to other people's minds, by the late Dr. Fagge. He said very truly that in the later stages of chronic granular kidney, where there has not previously been much œdema, you sometimes see a good deal of dropsy, which is not yet quite like renal dropsy because it affects the legs almost exclusively, and goes with gravity, and in other respects is more like cardiac than renal dropsy in its distribution. In these cases, he said, you will find, *post mortem*, the left ventricle much dilated as well as hypertrophied. I think he was perfectly right in saying that the dropsy which has not before been present, but comes on in the last stage of chronic Bright's disease, depends upon over-pressure in the arteries leading first to hypertrophy, and then to dilatation of the left side of the heart. After all, that is what we would expect. If obstruction starts in the lungs you at once get over-pressure in the right side of the heart, then in the veins, and then dropsy; if it start at the mitral valve you get over-pressure in the lungs, then in the right side of the heart, then in the veins, and then dropsy; if it start at the aortic valve, you get over-pressure and dilatation of the mitral, of the pulmonary capillaries, of the right side of the heart, then of the veins, and then dropsy. It is surely not strange that if there is obstruction in the systemic capillaries, there follows over-pressure, and high tension in the arteries, then in the left ventricle (first hypertrophy, and, afterwards, as it gets weaker and weaker, dilatation), then dilatation of the mitral, obstruction in the pulmonary capillaries, and finally over-pressure in the veins, and dropsy. That is, I believe, the true explanation of the dropsy in the last stage of chronic Bright's disease.

Lastly, there is the dropsy of anæmia, when chlorotic girls, and people with Addison's idiopathic anæmia, or with phthisis, get swelling about the ankles. I believe that this is chiefly, if not entirely, due to weakness of the heart. The pulse is feeble; the left ventricle is ill-nourished. It is not of the deep red colour that it ought to be; it suffers from want of hæmoglobin just as the muscles of the trunk do; and just as such a person has pale lips instead of bright red, pale instead of ruddy skin, pale blood instead of blood full of hæmoglobin, so he has pale cardiac muscle.

and feeble action of the heart. Accordingly the pressure in the arteries becomes low, in the veins high. A little exudation takes place. That is not noticed in the head and arms because it is soon carried away by its own weight; but it is noticed about the legs and ankles, where it settles by gravitation. It is, therefore, a form of cardiac dropsy.

In the treatment of cardiac dropsy you can do a great deal. You might say, on looking at such a case in the dead-house, "What is the good of putting drugs into the stomach, there is the disease and there it must remain, and the patient must die." Now, we know that is not the case. Although we cannot alter the condition of the mitral valve we can alter the effects it produces, and we can cure this cardiac dropsy as effectually as any other disease by drugs. Again and again you will see a patient swollen, water-logged, passing albumen, panting for breath, and at death's door; but, under proper treatment, at the end of a month or six weeks he will walk out without any dyspnoea, sleeping well, eating well, and, to all appearance, perfectly cured. That may happen twice or three times. One patient of mine, a cabman, came five times into hospital with cardiac dropsy during a period of seven years, with the same symptoms each time, and went out much better; he died at last; but a person in a better position in life, notwithstanding his disease, may live to a tolerably good age.

What are the methods of dealing with the disease? The abdomen and one side of the chest may be full of water: tap at once. When you have relief from the most urgent symptoms then give your diuretics and purge moderately. Use Digitalis; that is the sheet anchor in these cases. It is as useful and certain in its effects as Mercury in syphilis, or Quinine in ague. Get the patient out of bed, let him sleep in his chair, and prick his swollen legs.

As soon as you go beyond these primary cases the prognosis is not so good. When you have an old case of emphysema with dilated right side of the heart, and dropsy, you may treat it in just the same way. You seldom need to tap, for there is not much ascites; but you must give diuretics, and you will do good; still, Digitalis does not seem to act on the right side of the heart so well as on the left, and the effects will not be so striking. Cases in the last stage of Bright's disease are bad ones. There is organic disease of the kidney

which has reached almost its normal end; but still in these cases you may do great good by giving Digitalis. Do not give it in the early stages; but when the pulse is no longer hard, but soft and irregular, when the pulse is "renal" no longer, but cardiac, then Digitalis does good. Along with it give Tincture of Steel.

In anæmia the one drug to be used is the mixture or pill of Iron. Give plenty of it, and the dropsy will all disappear.

4. Lastly, we come to renal dropsy. The renal dropsy that comes on in a child that has lately had scarlet fever, and who is passing urine stained with blood, containing plenty of casts and full of albumen (tubal nephritis) is a very remarkable form of dropsy. That is the one to which Bright was first attracted, and which he identified with two other conditions—albuminuria during life, and disease of the kidneys after death. The mechanism of it is still much disputed. There is no extra pressure on the veins: in fact, it is the arteries that have the high pressure. I am inclined to look upon it as inflammatory. It is dropsy, no doubt; but it is not a passive dropsy. I believe it is an inflammatory oedema, like that of the lungs in Bright's disease. Whatever its nature, it is very different from the other forms of dropsy. You know it, apart from the examination of the urine and pulse, by its distribution. It is little governed by gravity; it affects the whole body (anasarca) and the serous cavities. It affects the lungs as well. The eyes—conjunctiva and eyelids—are affected, as also the face generally. The loins and genitals are involved; there is a "lumbar cushion," as Bright called it, the scrotum is swollen, and the prepuce often crumpled up, so that you wonder how the patient passes his water, though as a matter of fact it does not cause retention. I do not say you will never see these appearances in cardiac dropsy, but, as a rule, the genitals escape. If you see swollen face and scrotum and loins you may be almost always certain to find albumen.

The prognosis is probably the best of all. You can treat it almost as certainly as dropsy in mitral disease; and when you have cured your patient you have cured him entirely. Do not tap if you can avoid it. Avoid if you can even acupuncture, which is so useful in cardiac dropsy. You must not hesitate to do it if it is necessary; you must not let the patient die from dyspnoea when one side of his chest or belly is full of fluid; but do not tap if you can avoid it as it is apt to inflame

the skin—another evidence of the inflammatory nature of the condition. You cannot employ diuretics, since the kidneys are at fault, and you cannot act on them. Depend chiefly on the bowels and the skin. Purging is the first thing, diaphoretics next. Purge as briskly as you can. If necessary, if the patients are faint give brandy; but purge until you have free watery discharge from the bowels. The efficacy of this method is strikingly illustrated in people with such dropsy who have been struck down by cholera. The one condition has cured the other. Next, act on the skin. This is much more difficult, but you must try it. Give a hot air bath, or, what is much more comfortable and safer, a warm bath, and then wrap in blankets. The wet pack, Pilocarpine, and Acetate of Ammonia are also valuable diaphoretics. Do not give Mercury or Opium. By baths, packing, and diaphoretics, you will generally get the skin to act; and when you have the bowels and skin freely acting as well, you will find the dropsy begin to pass off. At the same time give Steel, Tincture of Steel, and let the patient eat little and drink plenty, of water so that the urine may be as abundant and dilute as possible.

These are the principal points that I wanted to bring before you to-day as to practically dealing with dropsy,—points that you will afterwards elaborate, and, I daresay, many of you correct and extend by subsequent practice. I hope you will find them useful in making a very important subject a little more definite and clear; having, at all events, certain clear principles by which to recognize the kind of dropsy, and the best treatment to follow. You will have to vary the treatment by your judgment in each case. You will often have to break the best rules to save your patient; but it is a help to have broad principles to guide you, even when you intelligently depart from rules.

Further Experience of Chloride of Ethyl in Neuralgias.—Dr. Edgar Gans, of Carlsbad, has used this method of local treatment with most brilliant results in the following cases:—1. Left-sided supraorbital neuralgia, cured by one application; 2. Lumbago, remained cured also after one application; 3. Several cases of hemicrania; 4. Pruritus scroti. The Chloride is applied in a fine stream by means of the bottles, with capillary opening, in which the substance is now sold.

Therapeut. Monatsch., 3, 1892.

A CLINICAL LECTURE ON A CASE OF MEDIAN CERVICAL FISTULA.

Delivered at Guy's Hospital, London, Nov. 29, 1893, by
ARTHUR DURHAM, F.R.C.S., etc.

I WISH to-day, Gentlemen, to tell you about an exceedingly interesting case we have now in the hospital; and to give you my reasons for the diagnosis which I have formed concerning it, after which I shall proceed to operate.

You know that in the process of development of the foetus various organs and structures, ducts and canals appear, fulfil their purpose, and then proceed to waste and disappear,—by a process of absorption in the case of the solid parts, by a process of closure and obliteration in the case of the ducts and canals. Sometimes, however, it happens that one or other of these foetal structures, organs, canals or ducts, persists through life, or is not merely represented by the little fibrous tissue that ought to remain, and does remain in almost all cases, but the organ, structure, duct or canal itself persists. Of course, it is somewhat changed from the condition in which it exercised its function during foetal life; but still it persists. When this takes place such remains of foetal structures are always liable to be troublesome, and may sometimes become dangerous. The degree of trouble they give rise to, and the danger they involve, will, of course, vary very much in accordance with the particular case and the particular structure concerned. If my diagnosis is correct, it is with respect to a persistent foetal structure that the condition with which we have to deal to-day has arisen.

In connection with the history of the case which I will now relate, very little need be said. As stated in the report, it is as follows:—

The patient is a man, æt. 20. When 4 years of age he had scarlet fever, immediately after which it was noticed that he had a small swelling situated immediately over the trachea, about three-quarters of an inch above the upper margin of the sternum. Two years subsequently, this was lanced. (Apparently, it had got larger and more troublesome, in consequence of which it was lanced.) Almost continuously since that time

there has been a discharge from it. This discharge has, however, caused the patient comparatively little inconvenience except when, as occasionally happened, the discharge ceased. (What is meant here is that the opening from which the discharge came got blocked up or healed over, the discharge accumulated in the space behind, thus giving rise to pain and discomfort.) The patient is a healthy-looking man, and the swelling appears to be his only trouble. It is described as being rounded in outline, about half-an-inch in diameter, and projecting about half-an-inch from the surface. It fluctuates. Its margins are thick and somewhat hardened. The surface of the skin over it is reddened. The summit of it is of rather a dusky hue; the skin dry and scaly. It is tender on pressure, and moves freely on the underlying structures. (On this latter point I would not be too sure. It is put down in the report in this way; but do not take that statement as from me.) It moves upwards when the patient swallows. From its upper margin there runs a hard cord-like structure which is attached above to the lower margin of the hyoid bone. Around especially the upper part of the swelling there is considerable induration of an irregular character.

That is the description of the case recorded the day before yesterday when the man came in. Yesterday the swelling opened, and some discharge took place. Microscopical examination showed that the discharge consists of pus corpuscles and a few red corpuscles. It was a curdy kind of pus that was discharged.

Here is the man, and you can see the swelling in the middle line. From the resistance it offers to my fingers as I grasp it between them, you will see a hard mass which I can trace distinctly up to the hyoid bone in front of the thyroid cartilage over which it is movable. Down below it does not seem to be quite so movable; that may be from adhesion to the trachea, due to an inflammatory process. The swelling, you will see, has burst and discharged.

What is it, then, we have here to deal with? At first sight, unless you made a careful examination, you might be inclined to suppose it an abscess in the front of the neck, or else a sebaceous cyst. It might be so. But in connection with this you have to notice, first, that the thing has been in existence ever since, or even before, the patient's birth; secondly, that the condition did not show

itself from the very first, but only after an attack of scarlet fever, when, according to the man's statement, it appeared as a rounded swelling; and in the next place, although that rounded swelling was punctured, and some matter evacuated so that he was, for the time being, relieved from the pain and inconvenience he suffered, it has never healed properly, but has remained open. The opening may have closed over a little, a scab may have formed, or even the edges of the wound may have united, but as soon as that happened the matter, or the exudation—call it what you will—has gone on forming and distending the sac which has again opened. Thus the thing has persisted, and in all probability will go on as long as the man lives, unless some measures are taken for getting rid of it.

Observe one point, a point which is very essential in making the diagnosis absolute, but one which we could not insist upon, or, at any rate, which I did not choose to insist upon. When I attempted to pass a probe the patient demurred on account of the pain it caused; but supposing I had persisted in the attempt it would probably have gone up in front of the structures of the neck as far, or nearly as far, as the hyoid bone; it would, in fact, have gone up in the middle of that hard cord, extending up to the hyoid bone. Consequently, my impression about the case is that it is one of those cases which some call median cervical fistula, or persistent thyroid duct. In the early stage of development there is a duct called the thyro-glossal duct, extending from what would be the upper part of the thyroid gland to the back of the tongue, where it ends in the foramen cæcum. In the early stage this canal or duct is open. It is in association with this that the isthmus of the thyroid body is said to be developed; and certainly it would appear to be in association with this, or with some persistence of this, that that extension upwards of the isthmus of the thyroid body that is sometimes found occurs—the pyramidal extension of the thyroid body; and it is probably in connection with this that those accessory thyroids are formed which sometimes are found in the middle line extending up from the thyroid.

Here I might just mention one of those strange coincidences which sometimes happen in the course of our practice and experience. It was only yesterday afternoon that Mr. Howse was

operating upon a case of thyroid disease for the removal or section of the median lobe or isthmus of the thyroid; and in that case there were these accessory thyroids and this pyramidal extension also, running straight up from the isthmus of the thyroid; in point of fact, illustrating one of those conditions associated with this thyroid duct of which I am speaking. In Mr. Howse's case the portion of the thyroid duct associated with the development of this part of the thyroid was very persistent; in my case it is the other portion of the bifurcation of the thyroid duct that has persisted—at least, so I judge—and which has given rise to the trouble we have here. It is a curious coincidence.

This thyroid duct, as a general rule, having fulfilled its function, passes away and disappears entirely as other ducts and canals of foetal life do. But, as I say, sometimes it persists. When it does so, I believe it remains in a quiescent state for a certain amount of time—we cannot say how long; but after a time, from some stimulating or exciting cause secreting action is set going. It may be the scarlet fever in this case had something to do with it. In one or two other cases of cervical fistula I have known an attack of scarlet fever render, or seem to render, that which before had seemed to be of very little trouble, a source of such trouble as to necessitate something being done. The exudation into the closed duct causes a swelling to appear, which may be the first thing to direct attention to the condition. Then the part inflames, and in this case it was opened. This is what generally happens in these cases. The inflamed and distended persistent duct does not generally open externally at first as some other cervical fistulae do, for example, those associated with the branchial clefts, which are generally open from the first. As a rule, these central median cervical fistulae associated with the thyroid duct are not open from the first, but only become so one, two, three, or four years after birth, or even later.

Once having happened the thing goes on. Secretion begins, the sac distends and eventually bursts, there may then occur temporary closure, but secretion again commences, followed by distension, and again there is discharge. But all the while this is going on in the interior of the duct some inflammatory action occurs above and around it; and this is an important fact to remember, because such an inflammatory process, by causing thickening

and hardening around it, makes it more adherent and closely connected with the surrounding structures, and more difficult to separate from them. It is just possible from the feeling this has in this case that there may be some new growth properly so-called present; but I am inclined to think that the induration and hardness one feels is merely of inflammatory origin.

Here let me remark that those remains of foetal life are very apt to behave badly, or, if not to behave badly themselves, to form the nests in which something of bad behaviour or malignant character is very prone to form. This being so it is very desirable in all cases where it is practicable to get rid entirely of those things that may prove a nuisance or the seats of serious evil.

What is to be done in these cases? One might freely open and scrape out the sac which is found in front of the lower part of the neck, but, that being done, though matters might be in a better condition for a time, very speedily, indeed, the same old story would be repeated, and we should have the same trouble as before. Only one thing, so far as I know, is effectual in these cases, namely, to dissect out and remove entirely all the abnormal structure. Such a process is not by any means always easy, and it is rendered more difficult by the inflammatory adhesions and thickening outside and around the part that has to be removed. It is not a thing to be turned out with the nail, we should be obliged to dissect it out, and seeing the structures on which, and amid which, it lies, such a process has to be conducted very carefully indeed. However, I do not despair of being able to accomplish this in this case. I propose by dissecting it out to remove all the abnormal structure we find, even though, as I have no doubt will be the case, on opening the sac freely, clearing out the contents and passing a probe upwards, we find it go nearly, if not quite, to the hyoid bone. However that may be, we shall trace it up as far as we can and dissect the part out.

What are our prospects of success? If we succeed in getting the thing entirely out right up to where the tube is obliterated we shall succeed in curing the patient no doubt; but if ever so small a portion of the lining membrane of the duct is left the probability is that it will become the starting point of further trouble. In these cases you must do it "all in all, or not at all." To be successful

you must get the whole out, or it will be of comparatively little use in getting a part out.

Any other method of treatment in these cases is probably futile. Over and over again injections have been made, cauterisations and other means have been tried; but always the trouble has been recurrent unless the whole thing has been completely removed; and that this is possible experience has shown. I myself have removed two or three. I have here a specimen which was taken from a little girl of six. In front of the neck, a little below the hyoid bone was a fistulous opening with a good deal of induration round it. A cord-like swelling extended up to the hyoid bone. It had been first noticed two years before, that is, when the patient was four years old, as a roundish swelling. This had been opened and some cyst wall had been cut away, but it had recurred, and some discharge kept coming away. In these circumstances I was called in to see her. It was clear that the only thing to do was to dissect out thoroughly, however far it might extend, the sinus or fistulous tract. I had some little difficulty in doing this. I dissected out right up behind the hyoid bone.

One point I am afraid I have omitted. The thyro-glossal duct, as I told you, passes from the upper part of the situation of the thyroid body to the foramen cæcum, which opens on the dorsum of the tongue posteriorly. When the hyoid bone is developing the thyro-glossal duct seems to be stopped, closing behind the hyoid bone, and becoming adherent or closely associated with the body of that structure. The portion above the hyoid bone leading up to the foramen cæcum then becomes obliterated. (Sometimes, by the way, cysts are formed in that,—dermoid cysts). The other part closes also, and, under normal circumstances, becomes obliterated: but if the thing persists the duct remains open, and extends as far down as before. You will thus see how it is, if it only extends normally as low as the isthmus of the thyroid body, that we find it sometimes as low as a point just above the sternum. You will readily understand that. The discharge which accumulates cannot escape through the skin at first, there being no opening. Extending in the direction of least resistance in accordance with gravitation it finds its way downwards, and the more the sac is distended the more is there extension downwards, and when inflammatory adhesions form round it it is pulled still further downwards; so that it gradu-

ally extends further than it was originally intended to open.

In the case of this child the fistula had not extended far down at the time. We dissected it all out, and sewed the parts together, after which it healed up entirely. On examining the specimen we found that a bristle could be passed through the top, so that I made a little further incision, and touched the upper end of the wound with the point of a cautery. Although this was done four years ago there has been no recurrence of the trouble.

Some years before that a little girl living in Long Lane, 10 years old, had been in hospital supposed to have had a dermoid cyst which was opened. It did not turn out very well; a fistulous opening having remained. When I saw her I came to the conclusion that nothing but complete extirpation of the cyst would lead to cure. I extirpated the cyst entirely but with very considerable difficulty. It went up not only to, but beyond the hyoid bone to the extent, I suppose, of about a quarter of an inch. I got the whole thing out, however, and the child got perfectly well.

We shall operate on the man you have seen and try to effect the same cure. We shall probably have some difficulty in getting the part nicely covered up; but I think we shall succeed. If we do not, however, we shall, at least, be quite free to confess our failure.

I wish, however, to call your attention to another class of fistulæ in the neck. The peculiar character of the fistulæ we have spoken of is that they are in the middle line. But we have fistulæ in the neck also which are connected with unclosed branchial clefts or fissures. These, however, are lateral, not median. Sometimes they are bilateral, one being present on each side. They are very troublesome things to have to do with. Some patients do not mind them very much, but others care a great deal, and would give anything to get the thing cured. A young lady with a little weeping spot on the side of her neck deems herself in a very unenviable position. In the case of a young man it matters less; but even he may feel it very disagreeable, particularly when the tract has got inflamed.

I have here a specimen, taken from the neck of a doctor's son, who had suffered from the condition all his life, having been born with it. A little weeping had always come from it. After an attack

of scarlet fever it had got very much inflamed, and there was a good deal of sloughing of the skin. On going to see him I passed a probe in at the opening, which was situated about three-quarters of an inch above the sterno-clavicular notch, and found that it travelled along the track as far as the cornu of the hyoid bone. I had no doubt at all as to the character of the fistula. We slit up the sinuses that had formed about it from the inflammatory action till we got on to the track of the fistula itself. When I got to the cornu of the hyoid bone I imagined I had got to the end of the matter, but was astonished to find the probe travel still further, and we had to follow with our dissection till the base of the styloid process was reached. To this it seemed to be attached, or in close connection with it. I cut it off there, and closed the wound up. The skin having been duly slit up over all the other sinuses resulting from diffused inflammation about the lower part of the neck they afterwards healed perfectly well, and although the operation was performed three years ago there has not been a drop of discharge since. For complete success this is the procedure that is necessary. The operation may be difficult; it may even be dangerous; but it will not be dangerous if you know what you are about, while if you go recklessly about it you may easily get into trouble.

What are the morals I wish to impress upon you. First, I want you to bear in mind the existence of these congenital or developmental persistent ducts, and the cysts and troublesome fistulæ to which they give rise. You have to recollect that there are those in the middle line associated with the thyro-glossal duct, and the lateral ones associated with unclosed branchial clefts or fissures. Recollect, too, that though these cases are rare, yet they do occur, and they may prove sources of great disappointment to you. A child is brought to you about four years old, with a small rounded swelling in the neck, which is thought to be very ugly. You say to the mother in the most cheerful way, "That is very easily got rid of. No doubt it is a sebaceous cyst or something of that kind." Or perhaps, if the skin is reddened over it, you say, "It is only an abscess that wants opening." You push your lancet in, and perhaps scrape out some of the contents and expect it to heal up nicely. The part contracts, and for a time it looks hopeful; but it does not heal. The mother comes again, complaining of a constant discharge from the

child's neck, and asking if nothing can be done to stop it. You perhaps inject it with Tincture of Iodine or Carbolic Acid and say it will be all right. But it does not come all right. The thing goes on; and the case involves a considerable amount of annoyance to you as well as to the parents. On the other hand, do not undertake the removal of these things with over-confidence. They often involve procedure of extreme difficulty; and the success of your treatment will bring you the more credit after having yourself acknowledged so much.

Just because these cases are rare it is important that you should be reminded of them. It is just your pronouncing correctly upon the exceptional case that you find occurring every now and then in your practice that will gain you the highest credit. Everyday cases are just everyday cases; but sometimes you will get a case that is not an everyday case; and it is well to be prepared for such.

A CLINICAL LECTURE

ON

LARYNGEAL TUBERCULOSIS.

Delivered at St. Thomas's Hospital, Dec. 5 and 12, 1893,

By **FELIX SEMON, M.D., F.R.C.P.,**

Physician for Diseases of the Throat, St. Thomas's Hospital.

GENTLEMEN,—The subject which we are going to discuss to-day is, both from its frequency and its severity, undoubtedly the most prominent and most important laryngeal disease. If you consider that, according to reliable general statistics, about every seventh human being dies from pulmonary consumption, whilst post-mortem records of various large hospitals prove that certainly not less than 20 per cent. of all patients dying from tuberculosis of the lungs have got some laryngeal complication, you will see that, broadly speaking, every thirty-fifth patient dying in the practice of a general practitioner is afflicted with the cruel disease which will form the subject of to-day's lecture; and no more need be said, I think, to show you the great importance of being thoroughly acquainted with the symptoms, the diagnosis, and the treatment of so prevalent and so serious an affection.

As just stated, laryngeal tuberculosis is a very frequent complication of pulmonary tuberculosis, and the first question we encounter here is this: Is the laryngeal form always a secondary complication, or does it also occur primarily? This question has been hotly contested for many years, and even now is not entirely solved, although the evidence in favour of an occasional primary occurrence of laryngeal tuberculosis is increasing. *A priori* there is, of course, no reason why the larynx should not occasionally be the seat of a primary tubercular lesion, just as well as the skin, the joints, the choroid, or any of the serous or mucous membranes. Indeed, in view of our modern conception of the bacillary origin of tubercular processes, and in view of the fact that the bacillus, in a very large proportion of cases, undoubtedly penetrates into the lungs together with the inspired air, it would even seem probable that such inhaled bacilli should settle in the primary air-ways before penetrating into the lungs, and that hence a primary laryngeal tuberculosis should even be a comparatively frequent disease. Facts, however, do not agree with this theory. Although undoubtedly in a comparatively large number of cases distinct tubercular lesions can be recognized by means of the laryngoscope in the larynx, whilst the most careful physical examination fails to detect any sign of pulmonary disease; nevertheless, it must not be forgotten that small foci, especially when centrally situated in the lungs, may for a while escape all our present means of investigation. And if we determine the frequency of primary laryngeal tuberculosis by the more reliable means of post-mortem examinations, we are justified in stating that the isolated occurrence of the disease in the larynx must be an event of the very greatest rarity indeed. I know of only a very few cases reported by reliable observers in which the post-mortem examination revealed the existence of undoubtedly tubercular lesions in the larynx whilst the lungs were perfectly intact; and, on the whole, you will not go wrong by assuming, when you find characteristically tubercular lesions in the larynx, that there must be pulmonary mischief, even if you cannot yet detect it by auscultation or percussion.

A very curious phenomenon with regard to the occurrence of laryngeal tuberculosis in the two sexes is the fact that it is much more frequent in men than in women, although the pulmonary manifestations of tuberculosis are about equally

frequent in the two sexes. It may be that occupation has something to do with this peculiar fact, although it hardly accounts for the great difference actually encountered. Thus Schäffer found a proportion of thirty-two male to seventeen female patients, and Heinze's results are similar. It also is a result of common observation, which you will often see corroborated in the out-patient's room, that the more severe forms of laryngeal tuberculosis are much more frequently met with amongst men than amongst women.

As to the causes of laryngeal complications of pulmonary tuberculosis, I need say but few words. We are now all agreed that the determining cause of the disease is the bacillus tuberculosis, that heredity plays a very great role in the disease, that insufficient food and clothing in early infancy, bad ventilation of rooms, and exposure play the roles of favouring factors, etc. It is, however, not quite clear what determines the localization of tubercular disease just in the larynx. People who use their voices professionally are by no means more frequently attacked than others who have a silent occupation. At the beginning of this century, and indeed up to the most recent times, the theory of auto-infection first raised by Louis, was a generally favoured one. It was assumed that the sputum from the lungs passing over possibly catarrhally affected or very anæmic membranes, in which erosions of the epithelium existed, infected these parts, and hence led to the occurrence of a laryngeal complication. It was especially urged in favour of this view that so frequently the same side of the chest and the throat were affected. I must confess that this argument has never appeared to me a plausible one, inasmuch as, surely, when a patient coughs and expectorates, the expectoration does not remain limited to that side of the larynx from the corresponding bronchus of which the expectoration comes; and I should look upon the coincidence of tubercular affections of one lung and the corresponding side of the larynx much more as an evidence of the affection being spread by means of the lymphatic vessels. At present the question is this: Does the laryngeal affection begin on the surface and penetrate into the lower tissues, or is the order of events just the opposite? In all probability there is something in both views; but the greater likelihood appears to be in favour of the view that the disease in a majority of cases begins on the surface and from there gradually penetrates into the deeper tissues.

We will now discuss the symptoms of laryngeal tuberculosis.

Usually the first laryngeal sign met with in tubercular persons is *Paræsthesia of the Throat*. The patient complains of all sorts of uncomfortable sensations,—pricking, a sense of constriction, soreness, occasionally some slight difficulty in swallowing, and a general feeling that something is wrong with the throat. Often enough you will find that a patient is at a loss how exactly to describe what he feels. Associated with these sensations is a tendency to more or less fatigue of the voice, slight hoarseness, even aphonia, with any vocal effort, and a slight dry hacking cough. If you examine the patient under such circumstances with the laryngoscope you will frequently be struck by a very considerable *anæmia* of the parts, an *anæmia* often strangely contrasting with the apparently normal complexion of the patient. This *anæmia*, as I have told you in a previous lecture, shows itself in the form of either a *general pallor* of the soft palate, pharynx, and larynx, in which particularly the epiglottis is conspicuous by its whiteness; or, on the other hand, in the form of what, at first sight, might almost appear to be *congestion*, but which, on closer inspection, is seen in reality to be injection of the capillary vessels, so marked that you can distinguish even the smallest branches on the intensely *anæmic* mucous membrane. In cases in which this *anæmia* is found coupled with such forms of *paræsthesia* as I have just described and with a more or less constant, slight, hacking cough, often enough also associated with feelings of general languor, the practitioner ought always to be suspicious of a commencing tubercular process, even if examination of the lungs should fail to detect any definite evidence of that disease, and even if no signs of constitutional disturbance,—emaciation, febrile temperature, and night perspirations,—should seem to lend colour to that suspicion. I have often enough been accused of being an alarmist when I have drawn attention to the possibility of these slight symptoms being the first warning signs of more grave impending mischief; but unfortunately in a large number of cases the subsequent development of events has proved the correctness of my fears. Needless to say, I do not by any means wish you to frighten people by telling them, when you find the complex of symptoms I have just described, that they are consumptive and bound to die; but I want you to be on your own

guard whenever—and this will be unfortunately too often—you come across cases of this sort. Pharyngeal and laryngeal *anæmia* are now, Gentlemen, generally acknowledged amongst laryngologists, and indeed amongst many general physicians, to be one of the most valuable premonitions of pulmonary tuberculosis.

Further on, the laryngeal complications of pulmonary tuberculosis may be fairly said to branch off in two different directions:—one set—the *specifically* tubercular ones—manifested by tubercular infiltration, the occurrence of miliary tubercles in these infiltrations, the gradual formation of ulcers, and finally, if the ulcerative process should exist long enough, lesions of the perichondrium and cartilages themselves; the others, more *accidental*, so to say, such as chronic laryngitis and paresis or paralysis of a vocal cord, the latter due either to implication of one of the recurrent laryngeal nerves in pleuritic thickening round the apex of an affected lung (in such cases it is usually the right cord which is affected, owing to the proximity of the right recurrent laryngeal to the inner aspect of the apex of the right lung); or to that peculiar waxy degeneration of the laryngeal muscles which was some years ago described by Eugen Fraenkel in Virchow's "Archiv."

We will now discuss these forms.

After a period of very varying length, during which either the initial symptoms above described may have existed without any evidence of pulmonary lesion, or, on the other hand, in which they have made their appearance in the midst of a florid pulmonary tuberculosis, the symptoms of graver laryngeal lesions begin to occur. Sometimes it is hoarseness, or loss of voice, sometimes difficulty in swallowing, sometimes pain, sometimes several of these symptoms together, in rarer cases difficulty of breathing, which are complained of. The occurrence of any of these symptoms will depend upon the form of the localization of the pathological process in the larynx. In cases of general tubercular infiltration the dysphagia and phonatory troubles will be most marked; if the infiltration be excessive, dyspnoea will be added; whilst pain usually begins to occur with the commencement of ulceration.

The tubercular infiltration may attack any part of the larynx. Very frequently it begins on the epiglottis and the arytenoid cartilages. Under such circumstances the latter are seen to be changed into two large pyriform or round bodies

representing two or three times the original size of the arytenoid eminences; whilst the epiglottis is also thickened to several times its natural size, and has lost its shape to a considerable extent, sometimes assuming a turban-like shape, and in other cases looking exactly like two semi-transparent sausages lying close to one another. The appearance of these swellings is a somewhat cedematous one. The œdema, however, looks denser than that observed there in the course of inflammatory diseases, or in those affections in which the œdema depends upon obstruction of the collateral circulation.

Very characteristic of the tubercular infiltration, as, indeed, of all stages of specifically tubercular laryngeal affections, is the intense *pallor* which, as you see, forms so conspicuous a diagnostic element in laryngeal tuberculosis. The *symmetry*, too, of the affection on both sides of the larynx, as a rule, is very marked.

Whilst the arytenoid cartilages and the epiglottis may be so characteristically disfigured that an experienced laryngologist will, at first glance, without having ever examined the patient's lungs or addressed to him any questions concerning his disease, be enabled to say that the case is one of laryngeal tuberculosis, the vocal cords, the ventricular bands, and the posterior wall of the larynx may, up till then, have entirely escaped. In other cases, however, the very reverse is taking place. Whilst the arytenoid cartilages and the epiglottis retain their shape, the ventricular bands are so enormously swollen as to completely cover the vocal cords. This condition often leads at an early period of the laryngeal complication to almost complete aphonia, and a similar condition is engendered if the mucous membrane in the inter-arytenoid fold should become tumefied, and on attempt at phonation be squeezed between the inner surfaces of the arytenoid cartilages preventing their approximation, and thus the phonatory closure of the glottis. The vocal cords, again, may, in other cases, first become tumefied, losing their brilliant lustre, and looking semi-transparent. This, also, will engender a certain degree of loss of voice.

After this infiltration, wherever appearing, has lasted for a very variable period, extending sometimes from a few days to several weeks, in other exceptional cases even to many months, a change takes place, due to the development of actual tubercles in the preceding infiltration. It is a

much ventilated question whether miliary tubercles can be seen with the laryngoscope in the larynx or not. Whilst a number of competent and reliable observers strongly maintain such a possibility, others, equally competent and experienced, absolutely deny it. I myself have seen, in a few instances, in the middle of a previously uniform, semi-transparent, greyish infiltration of the epiglottis, some tiny little yellowish specks which were quickly replaced by shallow ulcers; and I cannot help thinking that these must have been miliary tubercles, as they completely corresponded with what one sees on the post-mortem table in cases of general miliary tuberculosis of the lungs. Anyhow, it will be rather rare that one shall be able to actually see such an occurrence, on account of the brief vitality of these tubercles, the place of which is quickly taken by shallow ulcers. Whether the process is a downward or an upward one is, as I have already mentioned, not quite decided. Certain it is that these tubercles are found very close to the epithelial layer, and that their quantity decreases the further down you penetrate. In some very few cases the tubercles occur in the form of regular tubercular tumours which may be situated in any part of the larynx. Instances of this kind have been described by Schnitzler, John Mackenzie of Baltimore, Percy Kidd, and others. You all know the patient who was for some length of time in the hospital with a rather sessile, irregular tumour in the anterior commissure of the vocal cords, which rendered him completely aphonic, and the nature of which, from the nondescript appearance of the growth itself and from the absence of all other symptoms, it was difficult to determine. The age of the patient, however (about 52), and his sallow cachectic appearance made one naturally fear that it might be a malignant new growth. The tumour grew so slowly that it was at first impossible to remove any particle for purposes of microscopic examination. When I finally succeeded in removing a piece sufficiently large and submitted it to Mr. Shattock, I was not a little surprised to hear of the verdict that the tumour consisted of nothing but characteristic giant cells and masses of debris, tubercle bacilli being detected in the specimens. The rest of the tumour is now being actively treated with lactic acid, the patient has regained his voice to some extent, and there is no evidence of general tuberculosis whatever. Cases of this sort, needless to say, are likely to remain pathological curiosities.

Infinitely more frequent is the variety in which, after an eruption of tubercles in the previously-existing tubercular infiltration, a breakdown occurs. The ulcers thus resulting are at first very small, lenticular, shallow, with a yellowish lardaceous basis, and quickly coalesce, so that after a short time the parts affected get a generally worm-eaten appearance. Very characteristic is their pallor. If existing long enough the ulcers gradually extend, both in circumference and in depth. The mucosa and submucosa having been gradually eaten away, the perichondrium becomes affected, and general evidences of perichondritis appear. Finally, the cartilages themselves are involved in the pathological process. They become carious and necrotic, and may be expectorated whole or in pieces. The former fate not rarely overtakes one or both the arytenoid cartilages, a deep crateriform ulcer indicating their former situation. The epiglottis, on the other hand, is frequently very slowly partially or completely eaten away, so that nothing but a disfigured irregular stump indicates its former situation. By this time, of course, the subjective sufferings of the patient have enormously increased. Whilst spontaneous pain is not frequently complained of, the dysphagia, especially if the epiglottis, arytenoid cartilages, and interarytenoid fold are affected, may be so great that the patient, in addition to his other sufferings, becomes a victim to starvation because he is afraid of taking any nourishment, even the swallowing of his own saliva and expectoration being attended with excruciating pain. The cough, which forms in these stages one of the most distressing features of the illness, must in all probability be attributed to the concomitant pulmonary condition rather than to the laryngeal lesion, although in cases in which ulceration exists in the interarytenoid fold—and I may here incidentally remark that this spot seems to be a predilection seat for the development of tubercular ulceration—the denudation of the terminal branches of the superior laryngeal nerve resulting from the ulceration no doubt helps to contribute towards the irritation of this nerve, and thereby causes reflex cough. When matters have come to that pass the patient usually is totally aphonic, owing to ulceration of the vocal cords themselves. Meanwhile the pulmonary changes too have, as a rule, become so much advanced that with increasing weakness, pyrexia, repeated hæmorrhages, increasing difficulty in swallowing, and complete aphonia, death rapidly ensues. It

is, however, hardly necessary to say that the order of events here sketched is by no means universally the same. In a good many cases the ulceration first begins in the interarytenoid fold; and the first symptom complained of is great difficulty in swallowing, although hardly any infiltration or ulceration of the epiglottis or of the arytenoid cartilages exists. In other cases one or both the vocal cords become at first infiltrated, later on ulcerated, when more or less hoarseness or aphonia will be, for possibly a long while, the only signs of laryngeal mischief, without the existence of either pain or dysphagia.

Whilst the above changes represent what I have called before the *specifically* tubercular lesions of laryngeal tuberculosis, there are, as I mentioned at the beginning, some other laryngeal complications which cannot in the strict sense be called tubercular. These are first, chronic laryngeal catarrh; secondly, the paretic and paralytic complications.

Chronic laryngeal catarrh, and indeed chronic laryngitis, often really co-exist with pulmonary tuberculosis without the most experienced eye being able to detect any signs of actual tuberculosis in the larynx. It is probable that the bad nutrition of the parts so characteristically manifested by the persistent anæmia, forms a predisposing element towards concomitant laryngeal catarrh under such circumstances; and, indeed, one finds such a catarrh often enough in consumptive persons who have to use their voices a good deal. This chronic catarrh or inflammation may persist throughout the length of the pulmonary process without ever assuming specifically tubercular properties; or it may even be cured by appropriate local treatment; or, finally, it may pass over into the more characteristically tubercular lesions. I here wish to remind you of what I told you when discussing chronic laryngitis, viz., that an apparently simple chronic laryngitis, limited to *one* vocal cord only, must always raise the suspicion that the process is not merely a catarrhal one, but is in reality a local expression of some deeper constitutional mischief—tubercular, syphilitic, or malignant.

The second form of the non-specific laryngeal complications of pulmonary tuberculosis is paresis of both, or paralysis of one, vocal cord. The former, which usually appears at a very early period of the disease, must be looked upon, as I mentioned at the beginning of the lecture, either as a manifestation of simple weakness of the parts

or of waxy degeneration of the laryngeal muscles. In very rare cases the interarytenoid muscle appears to be singled out for this particular waxy degeneration. I well remember a case of chronic fibroid tuberculosis of the lungs which I had the opportunity of observing for a long time when I was clinical assistant at the Throat Hospital, and in which the most conspicuous symptom was complete aphonia. On laryngoscopic examination it was seen that the cords came well enough together on attempt at phonation for their whole anterior two-thirds, but that, corresponding to the cartilaginous part of the glottis, there remained a triangular, widely gaping opening, showing the existence of paralysis of the interarytenoid muscle. This paralysis resisted all forms of treatment. The case has been described by the late Sir Morell Mackenzie in his work, "The Diseases of the Throat and Nose." Another similar case I had the opportunity of observing here at the Throat Department for nearly two years. The patient was a sempstress, who suffered from a cavity in the left apex and consolidation of the right apex, and in whom complete loss of voice had been one of the first symptoms. The laryngoscopic appearances were quite identical with those seen in the first case. Every form of treatment failed, and I finally lost the patient from view after I had got for her admission into the Ventnor Hospital for Consumption.

Actual paralysis of one of the vocal cords is not rarely engendered. As I have said before, it is, from anatomical reasons, usually the right vocal cord which is thus affected. In exceptional cases, however, *e.g.*, when an enlarged tubercular mediastinal gland presses upon the left recurrent, or left pneumo-gastric nerve, paralysis of the left cord may also result.

The above described symptoms give you a general picture of the usual lesions of laryngeal tuberculosis, and will, in the great majority of cases, enable you to arrive at a definite diagnosis of the nature of the laryngeal symptoms under laryngoscopic examination. Not many diseases can be confounded with laryngeal tuberculosis. Amongst the most frequent sources of error I may mention chronic laryngitis, syphilis, malignant disease of the larynx, and lupus.

As to chronic laryngitis, you have already heard that it may accompany, in its usual form, a pulmonary tubercular process, but that it is most suspicious if it should be limited to one vocal cord

only. In doubtful cases you will, of course, apart from careful examination of the lungs, have recourse to microscopic and bacillary examination of the sputum and to observations of the temperature, to the question of emaciation, and indeed to the general constitutional symptoms of pulmonary tuberculosis.

As to syphilis, the diagnosis will occasionally offer greater difficulties, the more so as you must remember that there may be a coincidence of syphilis and tuberculosis in one and the same subject, and that the laryngeal affection may be syphilitic whilst the concomitant lung lesions are found to be undoubtedly tubercular. Again, it is quite possible that syphilis and tuberculosis may co-exist in the larynx itself, when the usual appearances of either affection will be more or less blurred. A cautious use of Iodide of Potassium under such circumstances will help in clearing up the diagnosis. As to the more ordinary cases, however, you are to remember (1) that whilst tubercular affections of the larynx are distinguished, as a rule, by the pallor of the affected parts, the lesions of syphilis are usually of a decidedly inflammatory character, and that whilst the phthisical ulcer shows no inflammatory reaction in its neighbourhood, the syphilitic is, as a rule, surrounded by an area of considerable congestion, often of œdema; (2) that the development of the tubercular ulcer is slow, that of the syphilitic very rapid; (3) that whilst the phthisical ulcerations, especially at the beginning, before they become confluent, are usually small, numerous, and situated on both sides of the larynx, the syphilitic ulcer, which from the very first, is, as a rule, much bigger than the tubercular ones, is usually solitary and unilateral, though occasionally a few distinct syphilitic ulcers may co-exist in the larynx; (4) that the tubercular ulcer is, as a rule, not nearly so deep and so sharply limited as the syphilitic one.

As to malignant disease, it will help you to remember that tubercular affections of the larynx are most frequently encountered in persons from 20 to 40 years of age, whilst malignant disease of the larynx, with the rarest of exceptions, only develops after the 40th year of life. Again, the fact that tubercular disease is usually developed on *both* sides of the larynx simultaneously, whilst malignant disease almost always starts from one side only is a good distinguishing feature. Finally,

* See the author's lecture on Syphilis of the Larynx "The Clinical Journal," 18th January, 1893.

the fact that in the great majority of cases malignant disease forms a much more developed actual tumour than laryngeal tuberculosis, and that in the former there is evidence, as a rule, of violent and often œdematous reaction in the neighbourhood, serves to distinguish it from the lesions of laryngeal tuberculosis. It must, however, be confessed that occasionally one encounters cases in which all these usual helps in differential diagnosis fail one, especially in the most advanced stages of both affections, in which the occurrence of secondary perichondritis may effectually mask the characteristic manifestations of either; and in such cases sometimes the question even as to the concomitance of pulmonary lesions does not suffice to elucidate the difficulty. I have observed in our hospital, and described in the Hospital Reports, a case in which the post-mortem examination left no doubt as to the co-existence of laryngeal carcinoma with tuberculosis; and I beg the more to draw your attention to this fact because it is even now still commonly supposed that malignant disease and tuberculosis exclude one another, a belief the fallacy of which has lately clearly been shown by a comprehensive work of Dr. Hanau, of Zurich, in which comparatively many observations have been collected proving that both affections may and actually do sometimes co-exist with one another.

Finally, as to lupus, which, of course, nowadays is considered as a mere modification of tubercular disease. It may be said that lupus of the larynx is a very rare affection, and that in the majority of cases the co-existence of the laryngeal affection with nasal, pharyngeal and cutaneous lupus will help you to clear up any doubt. Further, laryngeal lupus, as a rule, is not painful, and unless the ulceration is very much advanced does not give rise to dysphagia. Finally, its course in the larynx is much slower than that of a genuine tuberculosis; and even whilst ulceration occurs, the constant appearance of fresh nodules helps the careful observer soon to differentiate between it and genuine tuberculosis of the larynx.

(To be concluded).

THERAPEUTICAL NOTES AND FORMULÆ.

Surgical Kidney.—Dr. Leonard Weber, of New York, has found Creasote of very great value in cases of surgical kidney, or pyelonephritis,

arising from calculus or gonorrhœa, etc., in which, for any reason, surgical interference is contraindicated. This may be the case, for instance, when the other kidney is too diseased to do all the work, when the heart is too feeble to withstand the shock of operation, or when the patient is too old and feeble. The Creasote (beechwood tar) is given in doses of three to five minims three times a day, either in capsules or in alcoholic solution. He claims for it great power in overcoming the effects of purulent infection, and says that there is no necessity to increase the dose.—*N. Y. Med. Rec.*, 22, 1893.

Ascites from Cirrhotic Liver:—

R. Res. Copaib. ... 3iij
Spir. Rectif. ... 3j
Mucilag. Acac. ... 3ij
Aq. ... ad 3xij

M. Ft. mist. Sig.: One tablespoonful to be taken three times a day.

Bronchial Asthma:—

R. Pot. Iodidi ... gr. xxx
Pot. Chlor. ... 3j
Tr. Lobeliæ ... ℥ xxx
Syrup. Codeiæ ... 3j
Aq. Destil. ... ad 3vj

M. Ft. mist. Sig.: One teaspoonful to be taken every hour, until relief is obtained.

REVIEW.

The A.B.C. Pocket Diary and Memorandum Book. (Burroughs, Wellcome & Co., London. 1894.)

This is a very handy little book, of size convenient for the pocket, and contains, besides a diary of a week to a page, a great variety of information useful to medical men and pharmacists, and also lettered pages for addresses. The last part of the book consists of an alphabetical list of drugs manufactured by the publishers, including a large number which are their own specialities. As a pocket companion this little book is likely to have a large sale amongst practitioners, and we can honestly recommend it as such.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 10, 1894.

A LECTURE ON CHLOROSIS.

Delivered at the London Hospital, Dec. 5, 1893,

By **STEPHEN MAOKENZIE, M.D., F.R.C.P.**,
Physician to the Hospital.

GENTLEMEN,—I shall first read some notes of a case, not at any length, notes being always a little tedious and dull; but those of the case I shall read have been taken so clearly and expressed so well by Mr. Adams that it is a pleasure to read them.

Our patient is æt. 18. She was admitted from the out-patient department, complaining of dyspnoea after the slightest exertion, palpitation of the heart, pain in the stomach after food, and giddiness on stooping.

There is no family history of any hereditary disease. The patient was born in the East-end of London, where she has lived all her life. She went to school until she was 13 years of age; for the next nine months she was in service. When 14 she went into a confectionery business, where she used to make sweets. She is still in that business, but now washes bottles. Her hours are long, from eight to seven, with one hour off in the middle of the day; and she has to stand in a steamy atmosphere all the time. There is no history of alcoholism; the patient takes, as a rule, only one glass of beer in the week. There is no history of any previous illness.

Present illness. The patient was quite well until six months ago—June, 1893. The first thing she noticed was that when she had to walk quickly, or to do work requiring extra exertion, her breath became very short. At the same time she would suffer from palpitation of the heart and pain over the heart, which lasted for a day or two, and then went away. During the last four months, since August, the symptoms have become much worse. She has also had swelling of the legs at night, which disappeared by the morning; and pain and flatulence after food. The patient lies in bed, flat on the back, and seems quite comfortable. Respiration is easy, and she sleeps and eats well. The patient looks very white; there is very little colour in the lips, gums, and conjunctivæ. There is some pitting on pressure over the legs, especially well-marked over the ankle joint and along the anterior

border of the tibia on each side. The tongue is flabby, light in colour, and there is a small amount of white fur to be noticed in the centre. The teeth are very good, clean, and regular. The appetite is good. The bowels are very constipated, and there has been no action now for four days. The abdomen is somewhat flaccid; resonant on percussion, except over the left iliac fossa, where there is some comparative dulness. There is flatulence after food, but no abdominal pain or tenderness. The liver dulness is normal. The spleen is normal. The pulse is pretty regular, soft, and easily compressible, numbering 72 per minute. The cardiac dulness seems to be slightly increased to the right side, terminating at the centre of the sternum. It commences above at the upper border of the fourth rib. The apex beat is easily felt in the fifth intercostal space. On auscultation the first sound at the apex is reduplicated, and the second seems feeble. The first sound over the second left intercostal space is replaced by a soft blowing sound, which is stationary. The nervous system shows no signs of disease. The pupils are of medium size and react to light and accommodation. The plantar reflex and knee-jerks—obtained on each side—are normal. On ophthalmoscopic examination the discs are yellowish in colour, the outline extremely well marked. At the outside of each there is a small amount of black pigment. That is merely the choroidal margin. The arteries and veins are both pale in colour, and not easily distinguishable, except by their size. There is no anæsthesia or hyperæsthesia; and no evidence of enlargement of the lymphatic glands. The menstruation was regular until eighteen months ago, when it ceased for six months. During the last twelve months it has been very irregular, sometimes occurring at the end of two weeks, at others not for six. At present she has amenorrhœa for six weeks. The blood has been examined on different occasions. On the first occasion the corpuscles were 57 per cent., or 2,850,000 coloured corpuscles per cubic millimetre; and the hæmoglobin on that occasion was 30 per cent. On the second occasion the corpuscles were 64 per cent., or 3,200,000 per c.m., and the hæmoglobin 36 per cent. On the third examination the corpuscles were 66 per cent., or 3,300,000 per c.m., and the

hæmoglobin 38 per cent. On the fourth occasion the corpuscles were 73 per cent., and the hæmoglobin 42. On the fifth the corpuscles were 75 per cent., the hæmoglobin 40; and on the last occasion (to-day) the corpuscles are 82 per cent. and the hæmoglobin 40 per cent.

I now show you the patient before remarking on the case. All that you notice about her is that she is decidedly pale. As compared with those of a healthy person the colour of the hands, of the lips, mucous membranes, and conjunctivæ, all are very much below normal.

There is one point in the history of the case which I might introduce now, viz., that when the patient was first admitted there was a well-marked murmur over the pulmonary artery. A few days later, when I listened, it was not so distinct; it had faded; yesterday afternoon, when I happened to be in the ward, and visited her, it was much more easily detected.

There is nothing here I need ask you to verify for yourselves. All I can draw attention to as objective evidence in the patient is the pallor. The other facts will be brought out in the history and the remarks on the case.

In this case, Gentlemen, the most prominent feature—indeed the condition for which she was admitted into the hospital is the condition of anæmia; and it seemed to afford a good opportunity of discussing the clinical bearings of anæmia and the diagnosis of the particular forms.

The first thing we have to do in investigating a case of anæmia is to determine whether it is

- (a) Secondary, or symptomatic anæmia; or,
- (b) Primary, or idiopathic anæmia.

To do this we have to obtain a complete history of the case, and to examine the patient carefully all over, to ascertain any signs of a departure from health. By this means we are able to ascertain whether there is, or is not, any organic disease which has given rise to anæmia, or whether any signs of disease which may be present are simply due to the anæmia. In our case the history and physical signs reveal no organic disease which could have caused the anæmia, and such deviations from a healthy state as are present are explained by the anæmia which is present. We are, therefore, driven to the conclusion that the anæmia is the primary and essential feature of the case. There are two forms of primary or idiopathic anæmia:

1. Chlorosis. 2. Pernicious anæmia.

How are we to distinguish between these two? The distinction is made by the examination of the blood. In both there is a deficiency in the blood; but the deficiency differs in kind in the two cases. In chlorosis the hæmoglobin is deficient out of proportion to the corpuscles. In many cases the corpuscles are not sensibly reduced. You may see cases of chlorosis of considerable degree in which the corpuscles may quite equal the normal—90, 95, or 100. I have known cases of chlorosis with 102 and 105 per cent. of corpuscles; but in these cases the hæmoglobin was very markedly reduced. In our patient the corpuscular richness, you will see, is very notably reduced. When first examined it stood at 57 per cent., and afterwards, on successive occasions, 64, 65, 73, 75, and 82 per cent.; 57 per cent. is a very decided diminution. Other observers have found even greater reduction of corpuscles in cases of pure chlorosis. Laache has recorded a case of 48.8 per cent.; Coupland one of 41.8, Gowers one of 25 per cent., and Hayem one of 18.7 per cent. I only mention that to show that whilst the reduction of corpuscles is not the essential feature of the disease, it is present by no means infrequently, and that it may reach a very considerable degree. But what is more characteristic of chlorosis is that, with or without a reduction in the number of corpuscles, we have a reduction in the hæmoglobin, a reduction out of proportion to the corpuscular loss. In our case that is very pronounced.

In pernicious anæmia the great feature is the diminution of the corpuscles. The hæmoglobin is reduced to a certain extent, but not to a proportionate extent. In chlorosis, then, the main reduction is in the hæmoglobin; in pernicious anæmia it is in the corpuscles. You will gather this very well from some charts which I will pass round. The first one is a good example of simple anæmia. In all these charts the corpuscular richness is marked in black, the hæmoglobin richness in red. In the cases of chlorosis or chlorotic anæmia, the red line is always beneath the black one, whereas in pernicious anæmia the black line is below the red one. Where the corpuscles are markedly reduced as well as the hæmoglobin, as in our present case, some apply a special name to the condition, calling it pseudo-chlorosis or chloro-anæmia, indicating that the element of chlorosis is present, but that there is also a loss of corpuscles.

The first thing, then, in determining whether a case of idiopathic anæmia is one of chlorosis or

pernicious anæmia is to examine the blood and determine the number of corpuscles, the amount of hæmoglobin, and the relation of these to each other. Where the corpuscular richness is but little altered, with the hæmoglobin very much reduced, it is a case of chlorosis; where the corpuscles are greatly reduced, and the hæmoglobin is not proportionately lessened in quantity, the case is one probably of pernicious anæmia. There are only one or two other diseases where we find such profound reduction of corpuscular richness as in pernicious anæmia—in cases of severe accidental hæmorrhage, of hæmatemesis, of hæmoptysis, flooding in labour, or miscarriage, these may produce great blanching of the surface from temporary and profound anæmia. In these cases the corpuscular richness may fall exceedingly low. It is characteristic of anæmia due to hæmorrhage, that the corpuscles and the hæmoglobin go hand in hand; they are both reduced, and to about the same proportion. Where the corpuscular richness is below throughout, it is almost certainly either a case of pernicious anæmia or a case of cancer. In hæmorrhage the corpuscles and the hæmoglobin go pace by pace, but in cancer the corpuscular richness may sink to a very low ebb indeed—20, 22, 18, or more per cent.; and the hæmoglobin, in the individual corpuscle, as in pernicious anæmia, may be above the normal, so that cancer is a disease which has to be especially distinguished from pernicious anæmia.

The symptoms which are produced by anæmia are mainly in proportion to its degree. Most of them are independent of the exact causation. I have drawn out a table illustrating in a purely arbitrary but convenient way the method I have adopted for the estimation of the degrees of anæmia.

DEGREES OF ANÆMIA.

No. of Red Corpuscles per cubic millimetre.	Percentage of Red Corpuscles.
6,000,000	120
5,500,000	110
5,000,000	100
4,500,000	90
4,000,000	85 Anæmia commences
3,500,000	80 Moderate anæmia
3,000,000	75 Decided anæmia
2,500,000	60
2,000,000	50 Grave anæmia
1,500,000	40
1,000,000	30 Very grave anæmia
500,000	20
	10
	Fatal anæmia
0	0

Tendency to
hæmorrhages
proportion-
ate to degree
of corpuscu-
lar deficiency

The range in health, taking the two sexes together for the moment, of corpuscular richness, is from 85 per cent. to 105 or 110. Many healthy people, especially in the country, have more than 100 per cent. of corpuscles. In towns the tendency is for the corpuscular richness to be below 100; but below 85 per cent. anæmia commences. Between 85 and 75 we have what I call moderate anæmia. From 75 to 65 we have decided anæmia. At 50 per cent. we approach a much more important line of demarcation. Above 50 per cent. anæmia is comparatively harmless, and we rarely meet with the severer results of anæmia; but below 50 per cent., and in proportion to the degree below 50 per cent., we find all the grave consequences arising. All anæmia below 50 is grave, because all the grave consequences—hæmorrhage, dropsy, pyrexia, neuritis, etc., may arise. At 30 it becomes very serious indeed. A very large proportion of cases, however, which reach 30 or under 30 per cent. recover; but at 10 per cent. we reach the fatal limit. I believe, as a matter of fact, cases that have reached as low as 10 have recovered, but practically below 10, and actually below 7.5, anæmia is always fatal.

I have said that anæmia *per se* gives rise to certain tendencies. These are hæmorrhages, dropsy and pyrexia; and they may occur in any disease, however produced, when anæmia reaches the point I have named. I have published cases from different forms of hæmorrhage and other diseases with hæmorrhages which show that retinal hæmorrhages, and hæmorrhages into other parts, pyrexial attacks, and œdema, are really due to the degree of anæmia, irrespective of its cause. That is a point I attach much importance to; because by many retinal hæmorrhages are reckoned to be pathognomonic of pernicious anæmia. They are not. In cases of hæmatemesis, and hæmorrhage of any kind, these symptoms may occur.

Again, we have alteration in the shape and size of the blood corpuscles—what is known as poikilocytosis, the altered corpuscles being termed poikilocytes. It is thought by some that this modification of the form of the corpuscles is found entirely in cases of pernicious anæmia. Now that is not the case. I have here a drawing from the case of a woman suffering from chlorosis where there was a corpuscular richness of 85 per cent., quite to the normal standard, and hæmoglobin richness of 35 per cent.; and in it you will see just the same modification of shape as the diagram

of pernicious anæmia shows, and which is shown also in the diagram copied from Eichhorst. In a healthy subject the corpuscles are much more uniformly arranged and much more uniform in size than in pernicious anæmia. In pernicious anæmia the corpuscles undergo great modifications in form, and a good many megalocytes or very large corpuscles are present. In chlorosis the tendency is towards the presence of a large number of corpuscles smaller than normal. The average in health is from 6.5 to 8.5 or 9 μ ; but in chlorosis the corpuscles are often not more than 2.5 or 3 μ , and the largest are only from 3 to 6 μ in diameter.

From the characters of our patient's blood, then—from the deficiency of hæmoglobin—we conclude that she is suffering from chlorosis, and are able to differentiate it from a case of pernicious anæmia. As a matter of fact that is not a very difficult thing to do in the majority of cases. The difficulty arises in diagnosing pernicious anæmia from very severe forms of secondary anæmia. In cases of cancer, sometimes in cases of tuberculosis, in cases of concealed hæmorrhage, in cases of grave organic disease, it may be exceedingly difficult to say whether the anæmia is secondary or primary.

Having decided our patient has chlorosis we have to discuss the symptoms which are present. These I have narrated. You will remember that in Mr. Adams's report it is stated that the first thing the patient noticed was dyspnoea on exertion. This is extremely characteristic, and is very often the first "revealing symptom" of the anæmia. Why does a patient who is anæmic become short of breath? The answer is simple. There are fewer oxygen carriers in the blood, and these have to circulate more rapidly to fulfil the functions of respiration. Exertion makes a greater call on respiration, which therefore becomes hurried and difficult. Moreover, the heart—insufficiently supplied with blood, and that blood deficient in quality—is unable to keep pace with the respiration, so that there is a disturbance in the relationship between the respiration and the circulation. Thus dyspnoea is an extremely common symptom; and it is characterised by occurring on exertion and subsiding when that is over, and by there being no evidence of disease of the lungs to account for it—an extremely important matter in some cases.

Palpitation also occurs. Palpitation is the sense of disturbed and accelerated action of the heart. The heart may be quickened without any palpitation: by palpitation we understand the sense of

fluttering or beating which the patient experiences in the heart. Whenever the heart is in any difficulty it beats more quickly, and in some cases it does that without the patient being sensible of it. In other cases it gives rise to severe repeated beats, which becomes very oppressive, and of which the patient is very conscious. That is explained by the same conditions, the want of blood-supply in the muscle of the heart and the disturbance in the respiration. The struggle of the heart to do its work gives rise to pain in many cases, as in this.

Another symptom mentioned in the report was pain in the stomach after food. In many cases of chlorosis dyspepsia is present. All the organs, like the heart and the lungs, are badly supplied with blood, or the blood which is supplied to them is deficient in its vital properties, the consequence of which is that secretion is defective, and peristalsis is defective, the muscles of the stomach and intestine being all enfeebled—in fact there is said to be a want of tone.

We have also noted in this case, as in many others, the existence of constipation. Constipation is extremely frequently present, and is, no doubt, due to the same causes as those I have mentioned, namely, the want of tonicity in the musculature of the intestine, so that the fæces are not properly forced on. There is a want of vigour in the contractile forces. So much is the disturbance of digestion and defæcation associated with chlorosis that some would speak of a variety of chlorosis as "dyspeptic chlorosis." No doubt a division of that kind expresses a broad clinical fact. However, though chlorosis is sometimes present without dyspepsia, and we often have indigestion without chlorosis, still we must remember it is extremely frequent from that cause.

Then comes the giddiness. When the patient stooped she became giddy. Vertigo, tinnitus, and other functional disturbances of the brain are very common in cases of anæmia of any kind. In chlorosis we get it and in severe degrees of pernicious anæmia. This sometimes becomes very oppressive. Sometimes the patients have an exaltation of the special senses, dreading light and noise; sometimes they become very obtuse, the sight becomes defective, and the hearing fails. Perversions of the special senses are common in extreme degrees; but noises in the head and vertigo are more common in chlorosis, depending, no doubt, on the irregularity of circulation and the defective quality of the blood.

In this case, too, there was heard a systolic murmur over the pulmonary artery. This is a valuable symptom in these cases. It is present in the great majority of them. What is this systolic murmur which is so often heard in the pulmonary artery of patients suffering from anæmia? It would take a whole lecture to do anything like justice to the views which have been held on this subject. All one can say is that by most it is regarded as a functional symptom, *due*, that is to say, not to organic changes, but to the anæmia and disappearing *with* the anæmia. Sometimes this murmur is in the pulmonary artery, and heard over the second intercostal space, in others it is heard over the third left intercostal space, and is regarded by some as due to mitral regurgitation, of functional origin. Owing to feebleness of the ventricular muscles the valves do not close properly and regurgitation takes place from the ventricle into the auricle; but in this case there is less reason to invoke that view than the other. I believe it is really the altered quality of the blood which produces it.

Another feature of this case—a quite unimportant but an interesting one—is that there is reduplication of the first sound. It is stated by most writers that this is exceedingly rare; in my own experience it is very common and quite unimportant. I do not, as a rule, attach the least importance to it. I have watched cases in this hospital and in private practice for a period of ten or more years, cases coming from quite different causes on different occasions, and simply recorded in my notebooks “reduplication of the first sound,” in which nothing whatever has come of it. Reduplication of the first sound therefore, in very many cases may be entirely disregarded. I do not propose to stop to inquire into the mechanism.

In our case the patient has no ocular changes beyond the one which is most instructive; and that anyone who can use the ophthalmoscope can see—a vein and artery seen through almost perfectly transparent media, revealing the condition of the blood and blood-vessels. When a patient is suffering from anæmia, it is difficult to distinguish between arteries and veins. Mr. Adams says they are nearly indistinguishable as regards colour, they are of different size and distribution of course: but instead of the scarlet red of the arteries, and the blue red of the veins, the two are of very nearly the same tint.

In many cases of chlorosis we find a tendency

to neuritis. Mules and others have shown that in a large proportion of cases of chlorosis, very slight swelling of the optic nerve occurs. In severe cases of chlorosis undoubtedly this is very often present. I have seen many cases of the kind. In chlorosis there is very little tendency to retinal hæmorrhages. I said that all these changes—hæmorrhages into the retina and elsewhere, pyrexia, œdema, and other symptoms were more dependent on corpuscular deficiency than on hæmoglobin richness. On the other hand, in chlorosis there is more tendency to inflammation of the optic nerve and retina than there is in pernicious anæmia. Either may occur.

I pass round a drawing of an extremely marked case of hæmorrhage and neuritis in a case of pernicious anæmia in a little boy. Here is another drawing from a case of pernicious anæmia in a man, aged 18. This began as a comparatively mild case, but became one of the most severe cases of retinal hæmorrhages I have ever seen. Here is another from a fatal case of anæmia, showing the pallor of the arteries and veins and a single hæmorrhage. That was a very instructive case which was under my care for many years. He had repeated relapses. He several times appeared to recover, but eventually died of this disease. Here is a drawing of retinal hæmorrhages in a case of scurvy, in which there is a great loss of blood as in purpura, but in scurvy I think the hæmorrhages are not due to any specific lesion or the deprivation of any particular element of the blood, but purely and simply to the anæmia. This was another extremely characteristic case of pernicious anæmia, giving a good idea of the scattered hæmorrhages and the changes which take place in this disease. Here is a diagram of a case of scorbutic anæmia, not due to scurvy, but occurring in a patient who got most intense anæmia, proving fatal ultimately, and which, during treatment, did not yield to anti-scorbutic treatment. It illustrates the size which hæmorrhages may attain. That is the most severe case of retinal hæmorrhage I have ever seen.

What I want to emphasize is that in chlorosis the tendency is not so much to hæmorrhages as to slight degrees of papillitis and neuro-retinitis. Dr. Gowers has very properly drawn attention to the fact that slight degrees of papillitis or optic neuritis are especially liable to occur in patients who are the subjects of hypermetropia. All observers confirm this.

Our patient had a slight degree of dropsy in the feet. There was pitting on pressure over the lower extremities. This has entirely subsided with the rest in bed; and that is an extremely characteristic point. Slight degrees of oedema occur in simple and pernicious anæmia, but never become a general anasarca. In fatal cases of pernicious anæmia, we may find an excess of fluid in the serous cavities; but we never find a patient dropsical like a patient suffering from kidney disease; so that this is of importance where there is doubt on the subject.

The urine presents no important changes. Nor does it usually in chlorosis. It is usually pale, of natural quantity, and rather low in specific gravity. In pernicious anæmia it does sometimes present decided changes,—important as bearing on the nature of the disease; but it is right to know that the urine in simple chlorotic anæmia does not show any excessive pigmentation, because that is part of the argument as to its essential nature. It is not due to any excessive destruction of blood-corpuscles.

In this case the menstruation is deficient. It is so in the great majority of cases of chlorosis. The relationship of the disturbed menstruation to the anæmia is one which is often disputed. In many cases the patient is regular throughout the period of chlorosis; but in the majority of cases the menses are irregular, infrequent and deficient in quantity and in colour. But we find wide variations. Some have a great deal less than natural;—so much so that as a rule we may say amenorrhœa and chlorosis are associated together. In many of these cases leucorrhœa is present. The leucorrhœa is the only thing that could be urged as a drain upon the patient.

Chlorosis is most common in young women, especially between the ages of 14 and 24; but it is not uncommon in young men, in whom we may meet just the same kind of anæmia with just the same special features in the blood, and the accompanying symptoms just as pronounced. I dare say you are all familiar with the statement of Niemeyer which is often quoted, that all young girls who commence to menstruate before the development of the breasts and pubes, between 12 and 13, suffer severely from an aggravated form of chlorosis. There is no doubt that there is a general truth in this statement, though perhaps not quite to the full extent that he pushed it.

Chlorosis is occasionally hereditary. We often find instances where the mother has suffered from chlorosis in the same way, and it is very possibly

not so rarely congenital. It shows itself sometimes in childhood and may be a lifelong companion. Where it occurs in extreme degree very early in life it gives rise, as Virchow has shown, to what is known as hypoplasia,—that is to say, owing to the deficiency of blood, the blood-vessels are not called upon to undergo the developmental changes which they do in the healthy developing person. In consequence the blood-vessels themselves remain narrow; irregularities are often found in their branches, and the stature and build of the person is often very markedly implicated. This hypoplasia is also met with in another form of vascular disease—mitral stenosis, in which we have a physical condition somewhat allied to it. If the mitral orifice is very much contracted in early life the tendency will be for very little blood to get into the ventricle; the ventricle will send out a smaller charge of blood into the aorta, and so the blood-vessels will never attain full and complete development. In one case we have an altered quality or deficiency of blood; in the other we have a mechanical obstruction in the circulation; but the physical conditions are much the same as regards the arteries.

The character of the blood, the deficiency of hæmoglobin, its relative richness in other albuminoids, the fact that there is no drain on the patient to cause the anæmia, all go to show that chlorosis is a defective formation of blood. In pernicious anæmia, on the other hand, the evidence tends to show that it is not a defective formation of blood, but an excessive destruction of blood which brings about the anæmia. In chlorosis the character of the blood, the age at which it occurs, the absence of any proof of any destruction of blood or any drain on the system, all indicate that the essential nature of the disease is a defective hæmogenesis or defective functional activity of the blood-producing glands. The chief blood-producing organ, as you know, is the marrow of the bones. The spleen and lymphatic glands take very little share, and must be regarded as supplementary sources of blood formation rather than active ones. All the evidence, then, goes to show—and the evidence of treatment supports that view—that in chlorosis we have to deal with an inherited or acquired deficiency or feebleness of the blood-producing organs; and it is important to know that, because it ought to underlie the treatment which is to be adopted.

(To be concluded.)

A CLINICAL LECTURE ON LARYNGEAL TUBERCULOSIS.

Delivered at St. Thomas's Hospital, Dec. 5 and 12, 1893.

By FELIX SEMON, M.D., F.R.O.P.,

Physician for Diseases of the Throat, St. Thomas's Hospital.

(Concluded from p. 160.)

Coming now to the questions of prognosis and treatment I am glad to be able to say that in few diseases, perhaps, has such a general revolution of universal belief taken place within the last twelve years, with regard to both these questions, as in the disease now under discussion. Whilst the discussion on the treatment of laryngeal tuberculosis in the Laryngological Section of the International Congress of London, in 1881, in which a large number of the most distinguished physicians of both hemispheres took part, showed an almost general scepticism, nihilism, and hopelessness with regard to these two points—so much so that the statements of the few dissentients who ventured to assert that neither the prognosis was so absolutely hopeless as the adherents of the opposite view seemed to take for granted, nor the chances of treatment so absolutely futile, were received with general incredulity—nowadays the position of matters is, if anything, simply reversed, and some young enthusiasts go so far as to directly state that one could now with a great degree of probability promise patients who applied when suffering from the *early* stages of laryngeal tuberculosis that one could cure them!—Now, Gentlemen, I am most anxious to impress upon you that, having regard to the serious responsibility undertaken by the practitioner in this matter, the question as to curability should be put most clearly. Laryngeal tuberculosis, as you now know, is merely a local manifestation of a general infectious process; hence you can *never* promise that, even if you should be successful in arresting for the time being, one or another local manifestation of the general disease, you can thereby actually “cure” the latter!—Exaggerated promises of this kind can only lead towards altogether discrediting otherwise useful methods; and both with respect to the patient's welfare and your own reputation you cannot be careful enough in giving the patients and their friends clearly to understand that all you can do

in the present state of our knowledge is to *possibly arrest* some of these local manifestations!—If Koch's treatment had kept its original promises we should, of course, now be in quite a different position. That was a treatment calculated, as we were led to believe, to kill the *cause* of the general infectious diseases by destroying the bacillus without at the same time injuring the tissues of the body. Whenever that goal will have been achieved—and in my mind there can be no doubt that Koch's discovery of tuberculine, though unfortunately published absolutely prematurely, was a step in the right direction—we shall be in a position to actually promise a cure. At the present time we can only in a certain number of cases *arrest* the *local* manifestations. But whilst willingly admitting this, and, indeed, enjoining you not to commit yourselves to exaggerated statements, I wish to impress upon you equally strongly that even such an arrest of local manifestations is a great step in advance.

Even at the present day but too many practitioners when the painful and often excruciating symptoms of laryngeal tuberculosis occur, and when the patient sighs for relief from them, are inclined to console him with the cheap advice that he ought to “take care of his general health and let the larynx take care of itself.” Unfortunately, they omit to state *how* the patient is to take care of his general health if the terrible dysphagia which is engendered by the laryngeal manifestations in so many cases, prevents him from assimilating a sufficient quantity of food to counterbalance to some extent the ravages of the general disease! An attitude of this character cannot, I think, be sufficiently regretted. Of course, when phthisical patients in the last stages of ulcerative disease, complicated already by perichondritis, etc., seek relief, even the greatest expert cannot do more than slightly mitigate the pain by frequent local applications of anodynes in one form or another: but it ought to be the aim of the *general practitioner* to recognize the occurrence of a laryngeal complication of the pulmonary disease at so early a period that treatment can be undertaken with a reasonable chance of arresting these early manifestations. The relief experienced by the patient, and the general improvement following judicious local treatment under such circumstances are often enough equally pleasing and surprising; and in not a few instances one is nowadays lucky enough to actually arrest the illness, either for a time or

altogether. If I compare the number of my own patients in private practice suffering from laryngeal tuberculosis now living, whom I have treated in the manner to be presently described, with periods of my own former experience in which I, too, was under the influence of the general nihilism, governing for a long time the therapeutics of laryngeal tuberculosis, the difference in results is indeed startling. Of course, not all patients, even if applying at a sufficiently early period of the disease, will receive an equal amount of benefit. That depends (1) upon the *localisation of the mischief*, (2) upon its *form*, (3) upon the question of the *intensity of the general tubercular process*. But under all circumstances one ought to try to ascertain the state of matters in all cases at a sufficiently early period, because it is only in this way that a larger proportion may be saved than hitherto.

It is not my intention to trouble you with an enumeration of all the innumerable therapeutical propositions which have been and are nowadays enthusiastically made, and at first received, then disclaimed, and finally forgotten. I prefer to give you a practical outline of the method I have now followed during the last five years, during which, I certainly can say, my results have been more satisfactory than at any previous period of my experience.

So long as there is no demonstrable lesion in the larynx except the characteristic anæmia you will, of course, refrain from *any* intralaryngeal treatment, and treat the patient in accordance with the stage which the pulmonary disease may have reached. Should there be no signs of active mischief in the lungs, Iron and Arsenic preparations, Cod Liver Oil, selection of a suitable place of residence, and avoidance of sources of further complications, will at this period be your chief aim. Should there be a great tendency to weakness of the voice or actual paresis of the vocal cords, gentle external faradization over the larynx will act as a local tonic.

During the period of *infiltration* we are practically most powerless. It has been suggested by Moritz Schmidt and others that already at this stage the larynx should be treated surgically, *i.e.*, that the swollen parts should be scarified or incised energetically, and strong antiseptics rubbed into the scarified parts. But this treatment has never found general acceptance, and I have not been able to bring myself to adopt this procedure,

considering that this stage, without leading to considerable inconvenience, sometimes lasts for a very long time; and that, by artificially producing ulceration, you may actually hasten the whole tubercular process. Of course, should the infiltration, especially of the epiglottis and posterior parts of the larynx, be very great and cause great dysphagia, you will, in the first place, have to order a suitable, soft, non-irritating diet. You may give Cocaine pastilles, or spray with a weak Cocaine solution, previous to the patient's meals; or you may adopt the suggestion made by Dr. Norris Wolfenden, which is that the patient should learn to drink with his head well bent down and forwards over the edge of the bed, when, by an avoidance of the direct contact of the food with the swollen parts, swallowing is rendered much more easy.

It is with the beginning of actual ulceration that the real local treatment of laryngeal tuberculosis begins. And once more I would exhort you here not to allow this ulceration to become general before such treatment is adopted, inasmuch as the cases in which the whole larynx is practically one mass of ulceration nothing more can be done than just temporarily to assuage the patient's sufferings. So long, however, as the ulceration is limited, say, to the interarytenoid fold, or to a vocal cord or ventricular band, much can be done by the judicious employment of Lactic Acid. We owe the introduction of this drug into the therapeutics of laryngeal tuberculosis to Professor Hermann Krause, of Berlin. His attention had been aroused by the communication of Professor von Mosetig-Moorhof, of Vienna, who had extolled the merits of this drug in the treatment of tubercular disease of the joints. Krause utilized the suggestion for the larynx, and insisted—an important point to know—upon its use for tubercular ulceration of that part, not in the usual way of *gently swabbing* the larynx with a solution of the proper strength, but of treating the laryngeal ulcer *entirely in the same way as you would treat a tubercular joint* after laying it open; in other words, it is useless to merely dip a brush saturated in a solution of Lactic Acid into the tubercular larynx; and it is for the success of the treatment absolutely essential that the solution should be *firmly rubbed* into the ulcerated parts. This, of course, cannot be done by means of a common laryngeal brush. Krause has devised a form of curved forceps opening by means of a

spring-joint in the handle, and supplied with two projecting teeth on the inner surface of each of the blades of the instrument, which, on the latter being closed, just pass one another. A small plug of aseptic wool is wound round the laryngeal extremities of the instrument and between them, so that when these are closed the inner part of the plug is caught and firmly secured by the teeth.

The instrument is now ready after saturation with a solution of Lactic Acid to be introduced into the larynx. Previous to the application of the acid, however, the larynx ought several times to be sprayed or painted with a 20 per cent. solution of Cocaine, inasmuch as the Lactic Acid applications are often felt as extremely painful by the patients. And I must tell you here that it actually demands a sort of self-renunciation on the part of the surgeon to at first adopt this treatment; because it is a sad thing, indeed, to inflict additional pain upon those poor patients, many of whom at the time the treatment is commenced are already suffering severely. But you sometimes must be, as Hamlet says, "cruel to be kind;" and when after a few applications of the acid, your patient begins to tell you that his throat feels much better, that he can swallow much more easily, that he begins to enjoy his meals, and that altogether he is more hopeful, you will be reconciled to what at first may seem to you a somewhat barbarous proceeding on your part.

The solutions employed are from the strength of 20 to 80 per cent. The latter strength was the maximum recommended by the inventor of the method himself. Quite recently, however, Dr. Percy Kidd showed at a meeting of the Laryngological Society of London, a patient whose laryngeal ulcers he had treated with pure Lactic Acid, and with such excellent results, indeed, that no ulceration whatever remained behind. The arrest had already lasted for several months when the patient was shown. It will, however, only in a small minority of cases be necessary to employ the stronger solutions or the pure acid; and in the majority of cases you will find that 20 to 50 per cent. solutions are quite sufficient for your purpose. Needless to say the applications must always be made under the guidance of the laryngeal mirror. I should consider it absolutely unwarrantable for any man to attempt to apply such strong solutions of Lactic Acid as those mentioned to the larynx without being perfectly certain that he was actually rubbing

the acid into the base of the ulcer, and not perchance into the base of the tongue or even into the oesophagus!—A warning of this kind is not by any means superfluous. I remember an instance in which a patient suffering from ulcerative laryngeal tuberculosis was brought to me by a practitioner. After ascertaining the condition of matters and talking the case over with the medical attendant in private, I told him that I should suggest the employment of the Lactic Acid treatment, to which he readily assented. The gentleman, however, had, in the course of the examination, requested me to show him the patient's larynx in the laryngeal mirror, from which I naturally concluded that he was not himself conversant with the use of the laryngoscope. When he promised to carry out the treatment as suggested I could not help asking him whether he was used to making laryngoscopic examinations, to which he frankly replied in the negative. On asking him how under such circumstances he could carry out the treatment I had suggested he replied, "Oh, I shall manage." I then felt it my duty to strongly dissuade him from employing the Lactic Acid treatment as I was sure that only misery to the patient, and discredit to a valuable method could accrue if the latter were, however unintentionally, abused.

Never begin with a solution of greater strength than 20 per cent. If the patient bears that strength well, proceed on the next day, or, at the latest, on the second day, afterwards with a 30 per cent. solution, and gradually go up to a 50 per cent. solution, or, if necessary, to even stronger solutions. Needless to say, the applications will be all the more efficacious if *short* intervals be made between them. To apply a Lactic Acid solution once a week or once a fortnight is simply ridiculous. As soon as considerable improvement has been obtained and the base of the ulcer is clearing, or when there is even commencing cicatrization, prolong the intervals and return to weaker solutions. No hard and fast rules can be laid down with regard to the total number of applications to be made, the strength of the solutions to be employed, and the exact intervals desirable when the ulceration is mending; every case must stand on its own merits. Impress upon the patient before commencing treatment that you cannot promise a radical and lasting cure; and that whenever fresh sensations appear after an improvement, or even an arrest has been obtained, he ought to apply to

you *at once*. Nothing is sadder than to think how much good might have been done if the patient had only applied earlier.

By availing yourself of this method, you will in a good many cases have the pleasure of mitigating suffering, and adding to the span of valuable lives. In not a few cases you may actually have the satisfaction of arresting the process for good. I can now look back upon quite a number of patients who after having been treated once or several times in the manner described, are now free from the dreadful throat complication which threatened to curtail their lives.

In a number of cases, previous to adopting this treatment, it may be desirable or even necessary to scrape the base of the ulcers as originally recommended by Heryng, of Warsaw, exactly as you would scrape the granulating surfaces of a tubercular joint after it has been opened, previous to applying the Lactic Acid. This, however, is a proceeding which ought only to be adopted by men perfectly familiar with other intralaryngeal operations, because by scraping other parts than those intended, you are more likely to increase than to diminish the ulceration.

In cases, finally, in which the ulceration is too much advanced, and the general condition of the patient at too low an ebb to admit of any energetic treatment, you must be satisfied with blowing into the larynx, by means of a powder blower, soothing powders, such as :

Morph. Acet. ... gr. ½

Iodoform. Desodorat.

(Vel Iodol.)

Ac. Borac. ... āā gr. j

once or twice daily, or by giving the patient Cocaine lozenges, or letting him use a Cocaine spray previous to his meals. It will be found necessary, under such circumstances to increase the dose of the local anæsthetic after a certain interval; but in these cases you need not be too much afraid of giving larger doses, because when once this stage has been reached it can only be our aim to promote euthanasia.

Needless to say, all the measures so far recommended have only reference to the *local treatment* of the larynx; and it would be as unjustifiable, while employing these local measures, to neglect the *general* condition, as it would be to be satisfied with the latter, and to let the larynx take care of itself. In cases in which the patient is still strong enough that some hope may be entertained of

benefit from a change of climate, residence during the winter in the Riviera, in Madeira, the Canary Islands, Algiers, etc., may be deemed advisable: but whenever there is already laryngeal ulceration do not recommend any place, however suitable in itself, without at the same time introducing your patient to a practitioner resident in the selected locality whom you know to be competent to deal with the laryngeal complication, as otherwise the laryngeal disease is almost sure to go on unchecked, spontaneous cure of a once established actual tubercular ulceration of the larynx being amongst the greatest rarities ever recorded. I particularly warn you against sending patients afflicted with tubercular ulceration of the larynx to the Engadine. Experience has shown that however well high altitude may be adapted to the concomitant pulmonary disease, laryngeal ulcerations do not do well there; and you will find this emphasized by all the competent English and foreign practitioners resident at Davos or at St. Moritz.

Of the internal remedies recommended at various times for the treatment of pulmonary tuberculosis, I now, in the great majority of my cases, use but one, namely, Creasote in large doses, as recommended by the late Professor Sowerbrodt, of Breslau. For the success of this treatment it is absolutely essential (1) that the Creasote should be *absolutely pure*; and (2) that the drug be taken *immediately after meals*; because otherwise it is sure to cause indigestion and other gastric troubles. The Creasote is given in small gelatine capsules containing one minim of the drug. An addition of the same quantity of Cod Liver Oil might be made; but I have found that patients are much more apt to get indigestion when taking the combination than when taking the Creasote alone. You begin by giving the patient one capsule three times a day, immediately after meals, and at intervals of three or four days gradually increasing from one to five capsules each time, so that the patient finally takes fifteen minims of pure Creasote three times a day. No doubt a few patients have a sort of idiosyncrasy against the drug, and cannot take it at all; but these cases, fortunately, are excessively few; and I would particularly warn you against attributing every fleeting indigestion, from which tubercular patients so frequently suffer, to the use of the drug, and from at once discontinuing it. Only when a patient, after repeated attempts to take the capsules each time, soon begins to suffer

from indigestion, are you entitled to conclude that he cannot take the remedy. The use of the drug, combined with the local application of Lactic Acid in such cases in which this is required, has yielded such excellent results, not only in my own hands, but also in those of friends of mine who have adopted the same line of treatment,—with reference to this I would only draw your attention to the remarks made by my friend, Dr. Douglas Powell, in the new edition of his excellent "Diseases of the Lungs," p. 547—that I find it difficult to understand the recent onslaught made by Professor Stoerk against the employment of Creasote in large doses in the last number of Fränkel's Archiv. für Laryngologie. It may be that most of the patients applying to him have not taken the precautions recommended to you above, or that in many cases the latter had not been able to more than temporarily check the progress of the disease, and that they applied to him when in the last stages of their illness. Now, Gentlemen, I am far from saying that Creasote is anything like a *specific* for pulmonary or general tuberculosis; and, indeed, I lay stress upon my emphasizing this: but, on the other hand, I can honestly say that since I employed the combined method recommended, my results have been fortunately more satisfactory than they had been formerly, that I have a comparatively large number of patients now living in whom the laryngeal disease has been checked by the Lactic Acid treatment, whilst the pulmonary disease appears to have become arrested by the long continued use of Creasote. Some of these patients have now taken between 10,000 and 20,000 capsules, a fact speaking in itself eloquently enough for the duration of life under this treatment.

In conclusion, I can only recommend you so long as we have nothing better, to adopt the treatment I have described, and I hope that your endeavours may be crowned with successful results.

Pain in the Ear if of inflammatory origin may be much relieved by:—

R. Menthol in Powder	...	} part j of each
Camphor in Powder	...	
Liquid Vaseline	...	

M. Sig.: Several drops to be allowed to trickle into the ear frequently during the day.

(Dunn Medico.)

SOME IMPORTANT POINTS IN THE DIAGNOSIS AND TREATMENT OF MAMMARY CANCER.

Abstract of an Address delivered at the Watford Branch of the British Medical Association, Dec. 13, 1893, by

A. MARMADUKE SHEILD, M.B., F.R.C.S.

The origin of mammary cancer.—It is not too much to say that this all-important question is still shrouded in mystery. I shall this evening take the view that the earliest observed changes in mammary cancer are a morbid proliferation of the acinous epithelium, resulting in large nucleated cells, which never progress towards a higher type, and that these spread by local extension and by infection of neighbouring cell elements, with the same power of morbid proliferation. Thus cancer may be looked upon as an error of growth, as a divergence of the proper functional activity of the epithelium into morbid channels. Such a change is especially likely to occur at the menopause, when the secretion of milk ceases, and all sorts of irritations of the mammae from the pelvic generative organs are rife. There can be no doubt that irritations of the breast at this time may readily produce cancer. And these are especially sore or eczematous nipples, blows, and old inflammatory disturbances, or the sites of removal of previous innocent tumours.

As regards the parasitic theory of cancer, it is our duty to look upon this exceedingly important subject with an open mind. The life-history of sporozoa in the intestines of various animals is scarcely understood, and the presence of psorosperms or encysted sporozoa in cancer cells rest solely on microscopical evidence, and this is disputed by observers of great eminence. It is more convenient, for clinical purposes this evening, to consider cancer as an error of epithelial growth, for that, at all events, draws our very forcible attention to a preliminary cause of cancer—irritation of the tissues. The boundary line between chronic inflammation and cancer growth is a hard one to draw; and if one took a microscopical section of chronic mastitis from a person prominent in the world, one would probably find that pathologists would be very reticent in expressing positive opinions as to its cancerous nature or otherwise.

The practical rule we learn from all this is to attend carefully to the condition of the nipples, when sore or irritated. It is especially important to see that they are well protected in early adolescence, for the congenitally imperfect nipples often found are due, in my belief, to undue pressure of stays and corsets. Suckling is rendered difficult or impossible, and congestions and irritations lay the foundation of future chronic inflammatory changes and of cancer formation. Commonly a woman with cancer will tell us that she never could suckle with the affected breast, and such cases are only too likely to terminate in this terrible malady in persons of over forty years of age, especially when predisposed by hereditary taint.

Early symptoms of carcinoma.—First note that in examining a breast for cancer, we should never "pinch up" the breast substance between the fingers, but always lay the hand and fingers flat upon the breast substance. If we appreciate a distinct tumour against the flat surface of the fingers, be sure that such really exists. The characters of the formation in true carcinoma will be as follows:—

1. The tumour is of stony hardness, and of a somewhat square outline.
2. It infiltrates the breast substance, and does not move within it freely like an encapsuled innocent tumour.
3. The nipple is retracted, and the skin over the tumour dimpled.

The glands in the axilla may or may not be implicated. The patient may or may not be emaciated and worn-looking. The tumour may or may not be the site of the peculiar "lancinating stab" of cancer. All such signs as these are variable, and with such considerations as mental shock and grief, endemic influence or hereditary proclivity must be given that secondary position in forming an opinion which their extreme uncertainty affords. Discharge from the nipple is usually found in cysts, but bloody or ruby-coloured fluids may issue from the nipple in cancer; and here I show you a beautiful drawing of a hæmorrhagic cyst in the centre of a mass of scirrhus. There is nothing that exactly imitates the extreme hardness of scirrhus, and if cancer be not hard, it is then so rapidly growing as soon to dispel all doubts. The retraction of the nipple is a most fallacious sign. It is due to the drawing in of the ducts by the

fibrous contraction of the scirrhus. This may be exactly simulated by chronic inflammatory contraction, the puckering over an old abscess, a shrivelled cyst, or a fibro-adenomatous growth. The three conditions, where difficulties and pitfalls arise in practice regarding the diagnosis of early cancer are as follows:—

1. Chronic mastitis, with lobular indurations.
2. Deeply seated cyst.
3. Deeply seated abscess, tubercular or otherwise.

To-morrow morning anyone of us may be confronted by one of our patients, who is of the cancerous age, and has discovered a "hard lump" in the breast. We are naturally apt to share in her alarm, and are too prone to make up our minds that such a case must be cancer. Such deplorable errors have arisen from this attitude that we cannot be too much aware of the inherent difficulties which surround an early diagnosis in these cases. It is of the utmost importance that a definite opinion be early formed, and I would here draw forcible attention to the pernicious practice of waiting in these cases until the glands are enlarged, or prominent signs of cancer show themselves, the patient being meanwhile comforted with plasters and ointments. We are all too apt to fall in with the desires of patients to delay, and thus we prevent them really having the chance that operation affords.

The condition that gives us the greatest trouble in diagnosis, is undoubtedly chronic inflammatory induration.

This is frequently bilateral, the outline when felt with the flat hand is never definite like that of cancer, and it lacks the stony hardness of that disorder. Many of you will notice that I omit the question of local temperature in this all-important question. Cancer, especially bad cancer, is apt to be inflamed and hot, and chronic inflammation is so seldom unduly hot, unless irritated by applications, that local temperature cannot be looked upon as a distinguishing sign. Seeing that chronic mastitis often heralds cancer in the breast of an elderly woman, it is our duty to watch such cases with close attention. Some of them are syphilitic. Remembering this, it is my own practice, I give it you for what it is worth, to cover such a breast with a freshly prepared plaster of Mercury and Ammoniacum, while I give Iodide of Potassium. The breast is covered with "gamgee" tissue, and

firmly bandaged. At the end of three weeks, I again examine, and if the suspicious hardness should have increased under this treatment rather than diminished, I should counsel exploratory incision, with permission to remove the whole breast, in a patient of over forty years of age. Retraction of the nipple and puckering of the skin may be found in these cases, especially if there has been an old abscess.

Next, let us all remember that an apparently hard lump deep in the breast, may be a thick-walled cyst, or a chronic abscess.

I know of no absolutely certain methods of diagnosing these conditions except by cutting into them. They are best treated by dissecting the cyst or abscess freely out, and hence diagnosis and treatment can be effected by the same means.*

Time will not allow to enter into the diagnosis of cancer from such innocent tumours as adenoma. These are common in younger women, and mobility in the breast substance will distinguish them from malignant disease, more than any other one symptom.

We have thus got over the principal difficulties in the diagnosis of early cancer, for a short period of observation will settle the question in chronic mastitis, and an exploratory incision is urgently needed, in cases of "deeply seated lumps" which may be fluid or solid.

Conditions of the nipple precursory to cancer.—I have already urged the importance of this subject, and will only now draw your attention to the conditions of the nipple-eczema and the so-called Paget's disease as precursory to cancer. The latter, of which I show you a beautiful painting, is peculiarly red, raw-looking and florid, weeping with serous exudation, and devoid of projecting papillæ, vesicular exudation, or marked scab formation. Very similar conditions are seen on the glans penis in association with cancer there, and also on the skin of the scrotum. Scrapings of the cells, prepared with Liquor Potassæ, will show the so-called psorosperms. In persistent chronic eczema, or in this affection, any progressive induration beneath the nipple may certainly be looked upon as an extension of proliferating epithelium down the ducts, and true cancer formation, and early removal of the breast must be at once urged. It is these cases which often give the longest instances of immunity after operation.

* Cases were here related of errors in diagnosis in these three conditions, and illustrations of specimens handed round.

Treatment.—Complete excision of the breast is still recognized as the best treatment for mammary cancer, and we will commence this part of our subject by considering what cases are unfit or very unfavourable for operation. These may well be summed up as follows:—

1. Very fat women, especially when glycosuria complicates.
2. Extensive infiltration of the skin, especially with shotty nodules here and there interspersed.
3. Marked fixation of breast to pectoral muscle. This is to be ascertained by raising the arm to render the muscle tense. If manipulation of the breast be practised when the arm hangs by the side, the pectoral muscle will move with the breast in a very deceptive manner.
4. Extensive glandular implication, especially of the glands at the root of the neck.
5. Evidences of dissemination of the disease in other parts, especially the bones.
6. In the aged, and especially the spare and wrinkled, cancer will often form in the breast, and remain quiescent for years. Nay more! It will often shrink and shrivel up with the individual who bears it, never getting larger than a small walnut, and if ulcerated will scab over feebly, or discharge a scanty serous fluid. Such growths will lie dormant or retrogressing in the flat and shrivelled mammæ of old women for twelve or fourteen years. The glands are rarely affected, or the general health severely implicated, and the patient dies in the ordinary course of nature, from some cause not connected with the cancer. Such cases should seldom be submitted to operation.

Important points in the operation of excision.—Everyone thinks that they are able to operate on a cancerous breast, but to do the operation thoroughly is by no means simple. Let us first remember that the gland is not round like an orange, but often has irregular processes of tissue passing from its periphery, and these are peculiarly likely to be left behind. The skin should be much more freely removed than is usually the case, and early union of the wound should never be brought about at the expense of retaining infected skin; this is a most common error.

The question of opening the axilla is one of

vast importance. In spare women, where the condition of the glands is obvious, the axilla should be opened if the glands are markedly enlarged as compared with the opposite side, not otherwise. In fat women, it is essential to open the axilla, and so explore the glands with the finger; for otherwise, it may be impossible to know if they are enlarged or not. Opening the axilla, especially in inexperienced hands, increases the risk of the operation, in consequence of the liability of injury to the axillary vein, and the greater difficulty in keeping the wound aseptic. It is of much importance to remove the fascia and cellular tissue which overlies the greater pectoral, for here large lymphatic spaces exist, in direct communication with the axillary lymphatics. The preliminary cleansing of the skin is best effected by hot carbolized soap and water, followed by sponging with hot rectified spirit and water, and finally with a solution of Corrosive Sublimate (1 in 2000). The axilla should be most carefully cleansed by shaving, carbolic soap, and the perchloride lotion. The instruments and hands of the operators should be scrupulously clean, but powerful germicides should never be applied to the extensive cut surface. Good flushing with warm water seems sufficient to wash away clots, etc., and is followed by very satisfactory results. The drainage tube had still better be used as a general rule. In thin spare women it may be possible to dispense with it altogether. In such cases very firm pressure must be employed. When all such precautions as these are properly carried out, and especially when the axilla is opened, the operation of removal of the breast is not one to be looked upon as simple and easy.

Electricity in cancer of the breast.—The mysterious nature of electricity, and its supposed wonderful agency in controlling a fatal malady, surrounds this subject with much attraction to the suffering and credulous. For obvious reasons medical men of repute hardly like to meddle with electro-therapeutics at the present time. Our profession is ever narrow-minded regarding new remedies, and an attitude of antagonism is taken up towards electro-therapeutics which is quite unwarrantable. We must not confound electrolysis of a morbid growth, when heat is generated, acids and alkalies produced, and caustic action brought about, with the passage of powerful interrupted shocks through a tumour. It is this latter method which has been adopted in mammary carcinoma. It has been assumed, with much

ingenuity, that the cancer cells being of low vitality, would perish, while the cells of the tissues remain active. It is obvious that the only way this question can be settled, is by the faithful record of the results of all treated cases. I am aware that a considerable number of cases have, in the past three years, been submitted to electricity used in this way. The published results have been very scanty, and this must raise a doubt in our minds as to the success of the treatment. If once I was convinced that this method gave better results than excision, I should say it was our duty to adopt it, and I regret that in our large London hospitals, which number electrical experts among their staffs, this question has not been settled by the treatment of a definite number of cases, with a trustworthy report of the results. I may finally add that I have seen small nodules in the skin destroyed by the ordinary process of electrolysis, and that cases are rumoured of death having occurred from powerful interrupted shocks passed through the left breast—the cardiac action being probably inhibited. So the method is not altogether free from risk.

Treatment by escharotics.—It is quite possible to remove an entire breast by caustics, but the process is tedious and painful, and has little to recommend it. It is otherwise with open cancerous ulcers, with a tendency to fungation and foul discharge, these are often much benefited by caustics. The ulcer being well soaked with a 5 per cent. solution of Cocaine, a caustic application as Chloride of Zinc with Collodion, or Potassa Fusa, may be lightly applied. Ricord's paste, a mixture of Sulphuric Acid and Charcoal, is also useful, but should be very sparingly applied. The disintegrating tissues may be removed by the constant application of the balsamic preparations, especially Benzoin, when the Cocaine and Paste may be applied as before. A comparatively healthy surface may be thus obtained with little pain or risk, and the local and general relief is often remarkable. Personally I would avoid all pastes containing Arsenious Acid, on account of the uncertainty of their action, and the risk of general poisoning.

Galvano cautery.—There are still some surgeons who remove the breast by the incandescent wire loop. I have no experience of the method, which must be followed by much suppuration, and by a risk of secondary hæmorrhage. Yet we must remember that some of the older surgeons now living, deliberately state that long and profuse suppuration in a "breast case" is rather to be

encouraged, as more likely to eliminate all cancerous elements.

Question of prognosis.—Acute, rapidly growing, and brawny cancers will generally return in the axilla, and scar in about two years. Cases of early removal of a small cancerous nodule, when the axillary glands are not affected, will give long immunity. As a rule the more vascular and florid the patient, the earlier does the disease return.

Re-operation.—Recurrent nodules should always be extirpated, even again and again, so long as there is not extensive gland or systemic infection. Some very promising results have followed this practice, the subjects having lived for a great many years.

Cancer occurring in both mammae.—This is more common than is generally supposed, and the treatment is the same as when the cancer is unilateral, excepting that a very careful search should be made to see if there be dissemination of cancer generally throughout the body. In spare women both mammae may be removed at the same operation with good results. In cases when very large wounds have to be inflicted, it is perhaps better to do the operations separately. If after removal of one breast cancer should recur in the other, the remaining breast may be removed if there be no evidence of general systemic infection. All such cases present questions of difficulty, and no hard and fast line can be laid down for the guidance of the surgeon.

Some remarkable phenomena in the growth of cancer.—Cancer, especially recurrent cancer of the breast, seems to grow by "fits and starts," and will often remain stationary or even get smaller, the credit being given to some bogus remedy taken by the patient. This seems due to some interference with the vascular supply. I have seen and now relate one remarkable case, where three cancerous tuberos nodules, in a breast scar, almost entirely disappeared temporarily, under the bodily depression of a concomitant severe illness. So cancer may in very rare instances ulcerate and slough to such an extent as temporarily to destroy its own main bulk. The withering and puckering of cancer in the very aged has already been alluded to.

Some points in the treatment of cancer beyond the reach of operation.—It is a great error to say to persons with advanced cancer of the breast, "I can do nothing for you." Such a statement drives the patient into the hands of the charlatan, and

there is no part of our duty more important than the management of these distressing cases. The first indication is the relief of pain, and this is brought out by the judicious use of Morphia used hypodermically. Atropine may often be combined. To three drachms of the hypodermic injection of Acetate of Morphia, one grain of Sulphate of Atropia is added, and the injection dose is three minims, containing $\frac{1}{2}$ grain of Morphia, and $\frac{1}{6}$ grain of Atropine. The Atropia lessens the disagreeable effects of the Morphia on the bowels and digestion. Some few patients cannot tolerate Morphia; in these the Opium pipe may be instituted, or, failing this, various sedative agents of the pharmacopœia may be tried, one after the other, though Opium or its derivatives are our sheet anchors in these distressing cases. I have known the administration of Chloroform of the greatest use, especially when the bones or spine are secondarily affected. The nurse or relative may readily administer a few drops at a time on a pocket-handkerchief and thus a patient may be spared many of the mortal agonies attending the later stages of breast cancer. Locally, any lotion or ointment may contain Morphia, but I know of few more soothing applications to an incurable breast cancer than the old fashioned conium poultice. In the country this may be made from the fresh leaves; in the towns the freshly prepared succus conii may be mixed with an ordinary bread poultice, to the amount of $\frac{1}{2}$ oz. or more. The breast should be dressed under a spray of Sanitas, Eucalyptus, Sulphurous Acid, or Carbolic Acid.

Thus all offensive odours are obviated, to the comfort of all parties concerned. The parts may then be syringed with solutions of Chlorinated Soda (Liq. Sodæ Chlorinatæ 3ij, Aquæ Oj), or Iodine solution one drachm to the pint, or Sulphurous Acid solution (1 in 80). If there is much fungation and disintegration, painting the surface with pure liquefied Carbolic Acid is very beneficial, for it acts as a caustic, a deodorizer, and an anæsthetic. The applications are very numerous for these cases, and I merely wish to indicate the comfort that may be given to these patients by care in the application and selection of remedies. The diet should be mainly milk and fish, with farinaceous foods. Rich meat diet and wines merely serve to increase the growth of cancer, and it is a significant fact that patients under certain quack remedies, are at the same time adjured to abstain from meat. Œdema of the arm is best met by elevation and bandaging.

In some exceptional and desperate cases removal of the limb and shoulder may become necessary, thus clearing away the whole of the disease. I may finally mention that I have tried pyoktanin in those cases of incurable cancer, but I was quite unable to satisfy myself that it did any real good.

In such an extensive subject as we have considered this evening, it is impossible to even touch upon everything of importance, but I have indicated the main lines upon which our thoughts and discussions should run. It would be interesting to know of any cases of cancer of the breast following upon local irritation, any instances of long immunity after operation, and any cases of the effect of electrical treatment in undoubted cancer.

THERAPEUTICAL NOTES AND FORMULÆ.

Carbuncles.—Surgeon Bocarro brings forward a new modification in the treatment of carbuncles, which is as follows:—A sharp-pointed curved bistoury, held flat-wise, is thrust into the base of the tumour at any convenient spot, and pushed well home in the direction of the centre of the tumour and made to traverse it till its point, while yet under the skin, reaches a few lines beyond the opposite margin into the sound tissue. That this spot has been reached will be indicated by the feeling communicated to the hand of the absence of any resistance to the point of the knife from the brawny substance of the tumour. Then by a sawing motion of the knife first towards one side of the tumour and then towards the other, special care being taken that the point of the knife is all the time kept under the skin, and fairly beyond the circumferential zone of inflamed vessels, the whole mass of dead and dying tissues is thus detached from the living except at their marginal attachments. If the size of the tumour be greater than the area the bistoury could command at one sweep, from other points at the base of the tumour as many more such subcutaneous incisions should be made as would embrace the whole tumour. The bistoury is then put aside. Next, should there not already be a sufficiently large natural opening upon the central sloughing mass, by means of a scalpel, a deep incision, linear or crucial, as may be necessary, is made into it, wide enough to admit a finger of the left hand, and helped by this finger and hand the mass is tilted

up and cut away from its marginal attachments at a line corresponding with the subcutaneous incision already made. The object of this practice is not only to completely remove the central mass of dead matter, but also the marginal zone of inflamed tissues which, if retained, always gives rise to further inflammation and its consequences, unless thus carefully and promptly managed. When the whole sloughing surface has been removed, the open wound left should be well washed with strong Permanganate Solution, dusted over with Boracic Acid, and covered with lint smeared with Vaseline or simple ointment, and a firm bandage applied.

(*Ind. Med. Rec.*, 10, 1893.)

Trigeminal Neuralgia.—Dr. Glorieux warmly recommends the injections of Phosphate of Sodium in the treatment of trigeminal neuralgia. On the first day he injects ℥xv of a 2 p.c. solution, and gradually increases the dose till, on the fourth day, he administers ℥xliv, and then continues with this till complete recovery. The best solution to use seems to be one containing 3 or 4 p.c. of Alcohol in addition to the Phosphate.

(*Wiener Med. Pr.*, 26, 1893.)

Pneumonia.—Dr. J. Larraber, professor of children's diseases, Louisville, uses the following prescription invariably in pneumonia in children, and claims for it that it fulfils all the therapeutical indications in the disease.

R.	Sp. Æth. Nit.	℥iss
	Pot. Acet.	℥iss
	Liq. Am. Acet.	℥iij
	Aq. Camph.	℥iij

M. Ft. mist. Sig.: 3as for an adult, 3j for a child, every two hours.

In addition he strongly believes in Belladonna pushed to toxic limits in relieving the heart of undue strain.—(*Med. Rec.*)

Chronic Congestion of Larynx:—

R.	Creasote	℥iv
	Magnes. Carbonat.	℥ij
	Aq.	℥iiss

Sig.: A teaspoonful in a pint of boiling water as an inhalation.—(*Med. Rec.*)

Œdema of Glottis.—Three hypodermic injections of Pilocarpine at short intervals, removed all dangerous symptoms rapidly.—(*Mendoza.*)

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 17, 1894.

A LECTURE ON CHLOROSIS.

Delivered at the London Hospital, Dec. 5, 1893,
By **STEPHEN MACKENZIE, M.D., F.R.O.P.,**
Physician to the Hospital.

(Concluded from p. 166.)

One word as to prognosis. The prognosis is a particularly fortunate one in the majority of cases. In all cases where the disease has not reached a very high grade chlorosis yields to suitable treatment. Niemeyer's statement, however, that three boxes of 96 Blaud's pills will cure any case of chlorosis is a very misleading one. The great principle of treatment is not merely to treat actively, but by long continuance. Otherwise we often see disappointing results. Patients with chlorosis quickly improve but they are not easily cured. In order to secure a cure the treatment must be as protracted as the disease.

What is the treatment? Universal experience has shown that Iron is the essential thing which is wanted. The disease we have seen is a defective hæmogenesis, and we know that the Iron is markedly defective in the absence of hæmoglobin, of which Iron, although not the largest constituent, is the most important functionally. Iron is found by everybody to be the most successful and thoroughly efficient means of treating the disease. Sceptics have raised the question whether Iron does any good. They have shown that the greater part of it passes out by the bowel; and the blackening of the stools which is so constant a feature in those taking Iron, is a proof that a good deal does pass that way. Nevertheless in spite of what is wasted and passes out, Iron, and Iron alone, in many cases will cure the disease.

There are many ways in which it may be administered, and it is not necessary to insist on any one. The great principle of treatment in giving Iron is to give enough, almost to give the maximum dose the patient can take. Though I admit, and all admit who have studied the subject, that a great deal is wasted, the best results are got with the largest doses. It is one of those diseases where maximum doses should be given. The maximum doses are not anything heroic, of

course. Whether we give the Iron in the fluid or solid form is not material. I have mentioned several times the name of Blaud. Niemeyer said he owed his own professional reputation to his use of Blaud's pills, he having been a pupil of Blaud. Blaud's pills consist of Ferrous Carbonate, and are unquestionably an exceedingly efficient form. As usually prescribed there is an excessive amount of alkali in them—two equal parts of exsiccated Sulphate of Iron and Carbonate of Potash. Half that quantity of Carbonate of Potash is quite sufficient if any is given, and my own belief is that the alkali is quite unimportant, and we may give the Iron just as well without it as with it. The great thing is the Iron, whether you give the Blaud's pills in the old or the modified form. Another good form is the dried Sulphate of Iron, 5 grs., made into a pill with syrup; this is about three times the strength of Blaud's pill. The saccharated Carbonate of Iron, again, makes a very good pill; 5 grs. with syrup makes a very convenient size, and contains a suitable dose of the Iron. Of course there are Reduced Iron, Phosphate of Iron, Arseniate of Iron (which does not contain enough Iron to be of much use), and a great variety of preparations which are useful. The advantages of the pill form are several. In the first place, the dose is easily augmented without altering the formula. You begin with one or two pills three times a day; and it is then not much more difficult to take three pills. Pills are conveniently given with the meals, the Iron being then absorbed with the food. Again, when giving Iron for a long time, it does not blacken the teeth when given in pill form, as in the fluid form, causing disfigurement to the patient, and reproach to yourself.

Another very important element in the treatment of anæmia is rest. The patients want more Iron in their blood, and no calls to be made on their powers. In treating a case of chlorosis I always tell the patient to lie down for a couple of hours or so in the afternoon. Where it is a severe case and the patient is really bad, I keep her in bed for a week or two, or if not so bad as that I let the patient sit in bed for breakfast, get up later in the day, and lie down for an hour or two in the afternoon. I am sure that is a matter of great

importance; and the importance of it in all forms of anæmia has lately been emphasized by Dr. Frederick Taylor.* In our case that point is illustrated very well indeed. When the patient was admitted to the hospital she was kept in bed and put at once on an Iron pill; but when Mr. Kenneth Mackenzie, my house-physician, understood the case was going to be used for lecture, he took off the Iron, and gave the patient coloured water. In spite of that the corpuscular and hæmoglobin richness, except at the last estimation, have steadily improved. The hæmoglobin to-day is the same as before the last two examinations. The blood condition has generally improved. I attribute that improvement mainly to the rest in bed, and partly to the food which the patient has had. These two things together no doubt supply the explanation. And here I would mention that the food which we daily eat really contains all the Iron which is needed for ordinary purposes. The proof of that is to be found in the fact that living our ordinary lives we maintain the proper condition of blood-coloration. But in chlorosis we have to supplement that, because, as I say, the great thing is to make a profound impression, to knock loudly at the door of the blood-producing organs.

Other symptoms which you will have to deal with in many cases of chlorosis, are indigestion and constipation. In a very large proportion of cases these symptoms are present. Though you may combine the fluid form of Iron with antacids or acids, it is not easy to do so; but if you give your Iron in the form of pills, you can use other remedies for the digestive trouble. For instance, where we have a condition of atonic dyspepsia, an alkaline bitter mixture with *Nux Vomica* or *Strychnine* is of great service between the meals. It cleans the tongue, improves the appetite, stirs up the gastric secretion, and altogether makes the patient very much better. The Iron given with the meals is readily absorbed, and does its maximum amount of good.

The importance of aperients in connection with chlorosis is very strongly maintained by some. My late colleague, Sir Andrew Clark, in a paper written some few years ago, said that if he had to make choice of a remedy for chlorosis, and confine himself to one, it would be an aperient. That shows the value which many attach to it. With all deference to Sir Andrew Clark's great authority, I think myself the influence of constipation in

chlorosis has been very much over-estimated. I see a very great deal of constipation in perfectly healthy young people not the least anæmic, and plenty of cases of chlorosis where there is no constipation. Of course, one should correct it where it exists, whether the patient be chlorotic or otherwise; but I do not think, as some maintain, that the absorption products of the fæces get into the blood, and there exercise a destructive influence on the blood corpuscles. There is no evidence, as I have said, that there is destruction of the blood going on in chlorosis; therefore, I think it is a piece of hypothetical therapeutics.

Correct the constipation when it exists. There are several ways of doing so. Some prefer the vegetable aperients; and *Aloes* combined with *Myrrh* is an exceedingly time-honoured but very efficient one. Where there is menstrual deficiency it seems to stimulate not only the bowel, but the pelvic organs. Given with Iron I think the *Aloes* and *Myrrh* pill each night an excellent remedy. My own plan, however, is to give a saline aperient in the morning. I generally order equal parts of Sulphate of Sodium and Sulphate of Magnesium, and give the patient directions to take two drachms or as much as is necessary in half-a-tumblerful, or a tumblerful of water the first thing in the morning. I think this serves many useful purposes, one of the most important of which is that we secure a daily evacuation without any trouble, and sweep out of the bowel once a day all its refuse. If anæmia depends on products in the intestine which undergo absorption, as some say, by sweeping the channel clear once a day we get the maximum amount of good. The patient has had practically ten or twelve hours for digestion, and we may assume that all the food capable of that process has undergone it by this time, and that the purge sweeps away merely refuse matter, and so prepares the way for the food and the Iron. Thus all the conditions are met.

I think, Gentlemen, I have said practically all that is necessary to be said on the subject. The outlook in this case is a very hopeful one, the patient has shown such a ready tendency to recover. That individual peculiarity differs with each case. We shall now resume the Iron pills and saline aperient. In conclusion, let me repeat what I said about the importance of continuing the treatment in cases of chlorosis. Like everybody else I have seen dozens and dozens of cases in which the patient quickly improved under treat-

* "Practitioner," vol. li., p. 161.

ment, but which have relapsed for want of this. I should have told you, when you give a course of Iron to a patient, the first effect is to cause an increase of corpuscles, not an increase of hæmoglobin. As a rule, in treating a case of chlorosis you will get the hæmoglobin up to about 70, 72, or 75 per cent.; while you will have great difficulty in getting it up to 80, and still more to 85. Nearly all the cases that leave the hospital "cured" of chlorosis, have hæmoglobin richness of somewhere about 70 or 75 per cent., sometimes not above 65 per cent. That is my experience. Patients with corpuscular richness of 65 or 70 say they feel well; they eat and sleep well, they may have a passable colour, they feel capable of work and wish to return to their duties; but as soon as they are subjected to the strain that these involve and go back to their previous unfavourable conditions of life they quickly relapse. Therefore when dealing with a case of chlorosis never rest content until you have brought the patient to the maximum point, and earnestly urge your patients to go on using the Iron for two or three months after they are apparently cured. In that way many cases are prevented from relapsing.

A LECTURE

ON

THE TREATMENT OF FRACTURES.

Delivered in connection with the Post-Graduate Course,
at Middlesex Hospital, Nov. 30, 1893, by

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GENTLEMEN,—The subject we have to discuss to-night is the treatment of fractures; and you will understand at once that it will be impossible in the course of one short hour to deal with the whole question in full detail. I propose, therefore, to limit myself thus: first of all, to speak of the general principles which should govern us in the treatment of fractures in general, and then, having discussed that for the first half-hour, to devote the second half-hour to running over the details of the management of certain common and important fractures.

Before discussing the principles of treatment I want to make two preliminary remarks. The first is that the treatment of fractures is a far more important matter than is frequently believed. The popular opinion, at any rate, is that if anything short of a perfect result is obtained after a broken arm or a broken leg, it is proof positive of the incompetence of the surgeon. A flesh wound is regarded with horror, and its primary union with a linear scar is looked upon as a surgical triumph; and yet a broken arm is considered quite a common-place accident, and its perfect union with unimpaired outline of the bone, and unimpaired function of the limb, is regarded as a matter of course. Now you and I know right well that in the one case—the flesh wound healing by first intention—nature needs but very little assistance at the hands of the surgeon; but in the case of almost every fracture nature makes a sad mess of it unless she receives at every step the most intelligent and skilful aid at the hands of the surgeon. Nothing in the healing of soft parts is ever so disastrous or so far from the ideal as unaided nature sometimes obtains in the healing of a fracture.

There is no fracture which does not demand the greatest care on the part of the surgeon who treats it, and there are no cases which tax the resources of the surgeon more than do some cases of broken bones.

The second preliminary remark I want to make is this: that the union of a fracture is but a slightly specialised form of the union of any wounded tissue. There is no essential difference between the repair of a fracture and the repair of a wound of skin, muscle, fascia, or any other soft tissue of the body. The "callus" that we speak of as something peculiar, or to which, at any rate, we give a special name as if it were something peculiar—the callus which unites a fracture is exactly paralleled in the union of skin. In the case of a divided nerve or artery, so close is the parallel that the term "callus" has been recently applied to the uniting material of these divided structures.

The union of a fracture is not something *per se*, something quite different from everything else that we recognize, but proceeds upon exactly the same lines as the union of soft tissues. Hence, the great principles of treatment are the same in all cases of severed tissues, and the sutures and the dressing applied to a wound of the soft parts

merely become changed into the splints and bandages of a broken limb.

As I proceed, you will, I hope, see why it is I lay stress upon these two preliminary remarks.

In treating a fracture we must endeavour to accomplish in successive steps four great ends. These aims of treatment are in short—

Reduction of fragments.

Maintenance of the fragments in position.

Rest until repair is complete.

Restoration of function.

The first aim is the reduction of the fragments, the perfect replacement of every one of the fragments that has been displaced at the time of the fracture. The second aim is to maintain these fragments in their exact position until nature holds them firm in her healing cicatrix. The third is to maintain the parts at rest until they are so solidified by nature that they can resist the forces to which they are exposed, the forces of muscular traction and the action of gravity. The fourth object or aim on the part of the surgeon is to restore the complete function of the part. We must consider for a moment each of these four great aims in the treatment of fracture.

At the head of the list stands the REDUCTION OF THE FRAGMENTS; and I would very strongly impress upon you that this is the key of the whole situation. No matter what its position may be, the success of the treatment of any fracture, *quâ* fracture, depends primarily almost entirely upon the completeness with which we can restore the fragments, every one of them, to their proper position. This restoration should be done at once. You will say that is a very commonplace remark; but it is not quite so commonplace in practice as in our text-books. How often do we find a fracture put up imperfectly, and the surgeon contenting himself with the thought that he has put it up temporarily, as he says, and that when he takes it down he will put it up a little better. He does not get the fragments into quite their proper position at first, but he hopes that when the swelling has subsided or the pain in the part is a little less, and the patient more tolerant of his manipulations he will be able to get them into a better position. Now that is an absolutely fatal mistake. *The fragments should be replaced at once*; for several reasons; first of all, because at no time is it so easy to replace them as at first; secondly, because the outline of the part gets so obscured, first by hæmorrhage, then by cedematous

swelling, that if you do not seize the golden opportunity of the first hour or two,—moment or two, I might say,—you will have no time so favourable for the reduction of the fragments. There is a third very important reason why this should be done at once, and it lies here, that it is only when a fracture is completely reduced with the fragments exactly in position that it becomes easy, and, in some cases even practicable, to hold the bones in position. I shall have to mention immediately with reference to maintaining the bones in position, that, in nine cases out of ten, the great difficulty in doing that, lies in the imperfect reduction of the fragments.

The reduction, then, should be undertaken at once, and it ought to be exact. Pardon me if I emphasize that point; because it is really the secret of the successful treatment of fractures. If fragments are allowed to remain in their strayed position, they infallibly spoil the result. It is this displacement of fragments which leads finally to the grave interference with the function of the limb we sometimes see. What are the cases, for instance, of fracture of the humerus, in which paralysis of the musculo-spiral nerve results? Are they the cases in which the surgeon gets the two ends of the bones exactly end to end? Not at all. They are cases in which he has only partially replaced them, leaving at some point or other a jagged point which has caught the nerve. The same is true with regard to the joints which remain stiff after fracture. Take, for instance, fractures of the elbow joint. This is a particularly difficult group of fractures on all grounds; but one of the great reasons,—not the sole reason, but one of them—why we so frequently have stiffness after fracture about the elbow joint is that the fragments are not completely restored to their position, there is then an irregular scar in the bone which interferes with flexion or extension of the joint.

You will say it is very easy for me to stand here and tell you that in every case of fracture you are to reduce the fragments completely, and restore the bones as perfectly as possible to their proper contour, but that in actual practice it is a very difficult thing to do. No doubt it is. But what I want you to remember is, that its importance is so great that the difficulty must be overcome, and this is a case in which you should not fail to avail yourself of the advantage of anæsthetics. If you have difficulty in reducing the fragments of a broken bone you should not hesitate to give the

patient an anæsthetic, so as to be able to get the fragments back to their exact position. Think for a moment. If you have a difficult dislocation to treat do you hesitate for an instant to give the patient an anæsthetic, that you may be able completely and easily to reduce it? Why hesitate when the displacement is in the middle instead of at the end of the bone! If you have an irregular, uneven flesh wound, do you hesitate to give the patient an anæsthetic, so that you may be able to restore the muscles, aponeuroses, skin even, to their proper position, and fix them there by sutures? We know perfectly well that when a patient comes to us with a flesh wound our one chance of obtaining a linear scar and perfect union of the wound is to replace the parts completely and accurately at the time. So it is with fractures. If we fail at the first moment that a fracture comes under treatment to restore the fragments completely we shall fail all along the line—more or less, of course, in different cases. Do not hesitate, then, to use every means in your power to reduce these fragments completely. I am not at all sure that the time is far off when in certain cases of simple fracture the surgeon will feel it his duty, if he cannot otherwise reduce the fragments accurately, to cut down upon the fracture and restore the parts, just as we should in the case of a dislocated bone if we could not otherwise restore it to its proper position. In fracture of the patella this is often done.

The second aim is the *maintenance of the fragments in this proper position while nature consolidates the bone* by cicatrization, or, as I may call it, by forming a scar in it. Here, of course, we come at once upon the great subject of splints and bandages. I do not propose to enter into the enumeration or description of these splints, but only to point out to you one or two guiding principles in their application. The first of these is that the splints should be put on as soon as ever you have reduced the fracture. Do not wait until the swelling has come on and gone away before you put up the bone, but apply your apparatus at once. In the case of a wound of the soft parts you would not delay, but would at once bring the parts into their proper position; and, quite apart from the question of antiseptics, you would put on the dressing and hold the parts in place until the scar was firm enough to withstand the traction to which it is exposed. So with the broken bone; restore the fragments perfectly, and as soon as you possibly can, put on your retentive apparatus.

The second point is this,—that if you have got your bone completely reduced you will have but little difficulty in maintaining it in position. If you will think of any one of the difficult cases of fractures which you have seen—oblique fractures of the leg, for instance, which, in many cases, are exceedingly difficult,—you will, I believe, bear me out when I say that the difficulty has vanished as soon as you have completely reduced the displacement of the fragments. There is no secret for making the retention of a broken bone in its position easy, other than that; reduce it completely.

The third point I would impress upon you is this:—that the retentive apparatus should, as far as possible, be of such a nature that it keeps up an equable compression upon the whole length and upon the whole circumference of the injured member. Don't try to overcome difficulties by localized pressure, as by a pad under the heel, or over the front of the tibia, or on either side of a limb. If you rely on firm pressure at one point of the limb to press the bone back into position, you will fail, and you ought to fail. The secret of success lies in getting the retentive apparatus to ensheath the limb completely and the whole length of the limb, and to exert a gentle, equable compression on the whole surface. A great deal has been said about the advantage of plaster of Paris over wooden and other kinds of splints. A good deal of what has been said is, in my opinion, beside the point altogether. The real merit of plaster of Paris or any other form of moulded splint does not lie in the fact that it is put on once for all and takes but ten minutes to put on, nothing further being necessary for another six weeks; its real merit is that when properly applied it fits equably on the whole circumference and length of the limb, instead of the pressure being at certain parts of the limb only and irregular.

You cannot make a wooden splint adjust itself to the contour of the limb as a plaster of Paris splint can be made to do. That is the most important point of all that I have to say about this second great aim of maintaining the parts in their position. Of course, I need not remind you of the great cardinal rules that we all learnt years ago as to fixing both extremities of the broken bone, and not putting on the splints so tightly as to interfere with the circulation, nor yet too loosely. It is not often, indeed, that they are too loosely applied; but not unfrequently they are put on too

tightly. The main thing is to have a uniform compression of the whole limb.

The third aim of the treatment is to *keep the part at rest until the repair is so far advanced that the bone can withstand the forces to which it is exposed*—the forces, namely, of muscular traction on the one hand, and the action of gravity in walking, standing, etc., on the other. You will see at once that there is no absolute time-rule which can be laid down: for as these forces vary, the time must vary. Fractures of the lower limb will require to be kept at rest for a longer time than fractures of the upper limb; and fractures of the upper segment of either limb (the thigh and arm) will require to be kept at rest longer than fractures of the lower segment. Whatever differences of opinion there may be as to the treatment of the earlier stages, pretty well every one is agreed that a moulded splint, such as plaster of Paris, is the better thing to use later on. The generally accepted reason for this preference is, however, in my opinion a wrong one, namely, the ease with which it can be put on and its cheapness. The true reason is the equable fit of the splint; the uniform support of the whole length of the injured part.

The fourth aim of the surgeon should be to *secure the complete restoration of the function of the part*. If we fail in this, we fail altogether. It is no use telling a man that he has got an excellent union, strong as ever, of his broken tibia, if he is still lame; or a man who has broken his arm, that his humerus has united perfectly, when all the time he has musculo-spiral paralysis. The great aim to which the others are but the stepping-stones is, of course, the restoration of the function of the limb. Pardon me if I lay stress on this point, because it is just here we are apt to go wrong; we concentrate our thoughts so much upon the healing of the broken bone that we are apt to lose sight of the end for which that bone is united, namely, the restoration of the function of the injured part. Surgeons do themselves injustice in this matter; they leave their cases just short of being well, and the patients then go off to bone-setters, masseurs, and so on, and much credit is thereby lost. Do not forget that after the bone has firmly united with its contour completely restored, we have to make quite sure that the function of the injured limb is entirely restored.

What are the particular points to which we should especially pay attention? First, there is

the complete mobility of the joints concerned—not only the joints which may have been injured by the fracture, but also those fixed in the splints and bandages during the treatment of the injury. Complete restoration of the movement of the part should be aimed at.

The second great point is the restoration of the circulation in the part. Arterial thrombosis sometimes occurs; sometimes venous thrombosis. The rather abrupt cessation or removal of the support which the splints afford leave the part subject to cedematous swelling afterwards. We have not finished the treatment until we have relieved that, and got over these difficulties altogether.

The third great point requiring attention is the occurrence of paralysis; there must be no muscular paralysis. Putting it briefly, then, our patients must leave us without any limitation of movement, without cedema, and without paralysis.

And now we must turn to COMPOUND FRACTURES. What modifications in these therapeutical maxims are necessary when there is a wound leading down to the broken bone? None. It is well to be quite clear upon this point; the addition of a wound leading down to a fracture adds one condition and one only to a fracture, namely, the liability to an infection with pathogenic organisms. Acute inflammation of the bone, periosteum, and medulla, necrosis of the bone resulting from that, diffuse cellulitis in the limb, infective thrombosis of the vessels, particularly of veins, erysipelas, septicæmia, pyæmia—all these, which we recognize as grave complications of compound fracture, are traceable to the one great source—local infection with pathogenic organisms. We have then in a case of compound fracture one object to aim at in addition to the four we have been hitherto considering, but one of vastly greater importance than any or all of the others—*asepsis*. You have had a lecture this session, I believe, from the great apostle of aseptic surgery, Sir Joseph Lister. I would not, therefore, spend a moment speaking of any of the details of antiseptic or aseptic treatment; but I would just, in passing, remind you that the reduction of fragments in a compound fracture occupies exactly the same important position that it does in a simple fracture. Those of you who have ever put your finger into a compound comminuted fracture, know the temptation there is to think that because a fragment is lying loose it is therefore a mischievous thing, and should be removed. That may be a natural, but it is an

exceedingly unfortunate and evil tendency. The mere fact that a fracture is compound does not render it necessary or wise to remove a single fragment that has broken off, any more than it does in the case of a simple fracture, provided always you accomplish the first great aim of all—asepsis. As to the others, I am afraid I must not stop to run over them; but do remember that important fact.

About primary amputation in compound fractures, however, I must say one or two words. You remember the lists that used to be given in lectures and books of particular conditions that in a compound fracture necessitated primary amputation. That list has been very much reduced in late years; and if you have followed me thus far, you will at once see the justice of this statement that *there is no condition in a compound fracture which demands amputation which, if existing in a simple fracture, would not also demand primary amputation.* The dark shadow of pre-antiseptic surgery still hangs over us, and we are apt to think that great comminution, fracture into a joint, hæmorrhage, wound of a nerve, tearing across of one of the arteries of a limb, and a great extent of wound of the soft parts, must of necessity be followed by primary amputation. Let us get rid of that notion altogether. Not one of these conditions necessarily justifies amputation. Whenever you are face to face with the question of primary amputation in a case of compound fracture, ask yourselves the question, Supposing there were no wound at all in this case, should I perform a primary amputation? Unless you can answer that question in the affirmative do not amputate. What difference is there between an aseptic compound fracture, so far as comminution and its risks are concerned, and a simple fracture? No one would dream for one instant of taking off a man's leg because he had a simple comminuted fracture of the tibia, even if the tibia were crushed up into fragments, because he knows that, given an aseptic fracture, these fragments will be involved in the cicatrix, and will all be welded together into one solid bone. Comminution does not lead to necrosis. Who would think of amputating for simple fracture into a joint? No one. So in a compound fracture into a joint, provided you can secure asepsis, never think of amputating.

The matter really reduces itself to this: there are two great reasons why we should amputate primarily for compound fracture. The first is, *such in-*

terference with the circulation that the part below cannot be kept alive. That is not peculiar to compound fracture! it is conceivable in simple fracture. If, for example, you have compound fracture of the leg, and one tibial artery torn across, the posterior tibial artery cannot be felt at the ankle joint, but the dorsal artery of the foot can, on no account for that reason amputate primarily. A big artery torn across, pouring out a considerable amount of blood, will cause such a condition that primary amputation is rendered inevitable. That condition may occur in simple fracture, and demand amputation quite as much as in a compound fracture. Can the circulation be carried on? you ask yourself.

The second great reason for primary amputation is such injury to the soft parts that recovery with restoration of function is impossible; for example, in a compound fracture of the leg from direct injury where there has been such destruction of the muscles that even when the bone united the leg would be useless. In such a case an artificial limb would be infinitely better; and primary amputation is desirable. In the case, too, of compound fracture, or any fracture for that matter, where there is such injury to the main nerves of the part that they could not recover, and only a paralysed limb would result, you should amputate. These, however, are the only two circumstances in my opinion which justify primary amputation for compound fracture. But, you will say, Is amputation ever done for simple fractures? I should say, very rarely indeed; and yet it is not unfrequently done for compound fracture. Of course, Gentlemen, the explanation is this: if you compare simple and compound fractures together, compound fractures are the result of more severe, and especially more severe direct injury to the part than are simple fractures; and therefore the conditions necessitating amputation are more frequent in compound than in simple fractures. It is not because of the difference in their nature, but in the intensity of the fracturing force that the explanation of the fact is to be found. That is what I want to impress upon you. Never amputate simply because a given injury is "compound." You may wonder why I should labour this point. It is because all of us have seen limbs removed primarily for compound fracture which we know would nowadays be, and ought then to have been, saved. The actual cases of compound fracture calling for amputation are exceedingly few;

and the surgeon undertakes a very grave responsibility in ever removing a limb for compound fracture. I think if you will adopt my rule of asking yourself the question, Should I amputate if this were a simple fracture? and only to do it if you can give an affirmative answer, you will not go wrong.

Leaving now these general principles altogether, we come to the details of some special fractures; and the fractures I should like to run over are those of the skull, the jaw, the clavicle, and compound fractures of the fingers.

Fractures of the skull are exceedingly common and very important; and what I have to say with reference to them will be of the fracture *quâ* fracture only, with not a word about any intracranial complication, because that would introduce too vast a subject. There are two things to be done for fracture of the skull. First, when compound,—and the great majority of them are,—render them aseptic. Compound fractures of the vault of the skull have the peculiarity that often they are mere linear cracks when the surgeon sees them, or bits of bone have been bent in and possibly have sprung back again, or the inner table has been broken up into smaller fragments which are driven downwards towards the membranes. In such circumstances in the fine fissure which results some dirty septic matter from the outside is frequently caught. It is quite a common thing for dirty bits of hair, for instance, to be driven into a fissured fracture of the skull. That is why we have to use very special means to render these aseptic. In such a case as that do not content yourself with pulling out the hairs with a forceps or anything of that kind. Either put on a trephine, if the fracture is extensive enough to require that, or with a gouge or very sharp chisel cut away a little groove of the outer table of the bone on either side of the fracture and lift out the edges of the fracture with the dirt, hair, or bit of hat, whatever it may be, that is held between the edges. Explore these compound fractures very carefully, remembering the extreme elasticity of the skull and the great liability to the inclusion in the linear fracture (I am not speaking of great depressed fractures) of hair and other infective particles which may afterwards give rise to the most serious complications. For fractures of the vault of the skull do give rise to the most serious complications. The big veins in the diploe readily become thrombosed, and if a septic thrombus forms there it may spread,

usually into a sinus inside, from thence into a cerebral vein, and possibly lead to cerebral abscess or leptomeningitis, so that you must be very careful to render these fractures aseptic.

The second point in fractures of the vault of the skull to be attended to is the removal, or, at any rate, the elevation of all fragments depressed on to the membranes or brain. You must get rid of the distinction that used to be drawn between simple and compound fractures. A simple fracture is but an aseptic wound, if I may say so; a compound fracture is but a simple fracture with the added danger of infection with pathogenic organisms; and provided you make it completely aseptic there is really no essential difference between the two. So in these head injuries it does not matter whether a man with a depressed fracture has got a wound over it in the scalp or not; if he has fragments of the bone driven into the dura mater and into the brain, they should be lifted up, and that will often—though not invariably—involve the use of the trephine. If only the wound can be kept aseptic the fragments can be replaced and enclosed in the scar that will result in the bone. We do not now throw away the disc of bone we remove with the crown of the trephine; we carefully preserve it, and restore it to its position, so as to leave no aperture in the skull.

In fracture of the base of the skull, with displacement, we can do nothing; it is beyond our reach altogether; and our duty is limited to rendering and keeping the part aseptic. Take, for example, a case of fracture of the middle fossa with hæmorrhage and escape of cerebro-spinal fluid from the ear. The same care must be taken to secure asepsis as in the case of a broken leg, or any other compound fracture. You should do this by very thorough and careful cleansing with an antiseptic—either Carbolic lotion, 1 in 20, followed by 1 in 40, or Bichloride of Mercury solution, 1 in 2,000. You should syringe this solution into the auditory meatus frequently, so as to get it as clean as you possibly can. You should, using all gentleness, rub with the lotion the surface below and around the ear, oftentimes engrained with dirt, the back of the head, shaving the hair from a sufficient area, after which you should put on a proper antiseptic dressing as carefully as in any other compound fracture.

Fractures of the jaw are very common injuries and often give rise to a good deal of difficulty. I want to speak of one treatment only which you all

can and ought to learn to carry out. It is the treatment first of all instituted by an old student of this hospital, Mr. Hammond. At the time of the Franco-German war he was in Paris, and saw a good many cases of difficult fractures of the lower jaw that were unsatisfactorily dealt with by the horse-shoe splint and other means. So he set about devising a treatment at the same time simple and efficient. He is a dentist; but his treatment is within the powers of any surgeon. As first introduced, his treatment involved the making of a cast of the jaw and fitting on a splint to that; but it can perfectly well be done without that detail. It consists of a ring of iron wire, tinned on the surface partly for cleanliness, partly that it may be easily seen. It is of that size commonly called "bird-cage wire." With this an "interdental splint" is made. (Mr. Gould here showed a splint that had been fitted on to a jaw by his colleague, Mr. Storer Bennett, and then himself went through the various steps of the process). The result of that treatment for fracture of the jaw is that the patient does not require any external bandage at all, nothing but the splint, which is left on for three weeks. The movement of the jaw is not limited. Talking can be carried on just as well as before, and he can also eat; and those of you who have treated fractured jaw by the ordinary horse-shoe splint know full well that one of the grave disadvantages—one of them, but not the only one, by any means—is that it interferes with the patient taking food. This plan does not interfere with the patient taking ordinary food. To apply this splint is not a difficult thing which only dentists can do, but is quite practicable for the ordinary surgeon.

Fracture of the clavicle. In this fracture the displacement that results is that the outer fragment falls down by the weight of the limb, whilst by muscular traction it is drawn inwards and forwards. There are very many ways of treating fractured clavicle, and from that fact you may be sure that there is some special difficulty in its treatment. There is no doubt difficulty in treating fractured clavicle, because we have not only to deal with the force of gravity, but with the very powerful muscles passing from the trunk to the upper limb, and we have no efficient means of opposing that inward traction of the muscles except by the patient lying perfectly flat in his bed; then we get splendid results, but that is, as a rule, objected to by the patient. One common

method is to put a pad in the axilla so as to force out the bone. I want to caution you against that. It never does any good. That is an easy point to demonstrate. This bandage (an ordinary roller) you will acknowledge, is not an unduly large or firm pad if you were going to use a pad to force out the shoulder; but if I put it in my axilla and draw the arm to the side, as it would be in the case of a fractured clavicle, I am perfectly certain that my radial pulse is stopped. It is impossible to put a pad in the axilla of such a size and firmness as to force out the shoulder against the pull of the pectoral muscles, the latissimus dorsi and the rhomboids, without first compressing the axillary vessels and nerves against the humerus. Never use a pad in the axilla for forcing out the shoulder; but there is a way of getting the shoulder out, and, so far as I know, only one.

I said just now the most successful way of treating a fractured clavicle is by letting the patient lie on his back; and if the patient happens to be a young lady with no irresistible calls on her time, that method should be adopted. But this is not always practicable. In that position the weight of the shoulders draws them back, and you cannot put your shoulders back without drawing them out. *The only way to get the shoulder out is by drawing it back.* If you can only do that you correct the outward displacement as well. The simplest way of doing it is by what is known as Sayre's method. I want to do this on the subject before you, because there is one little point that frequently trips up the surgeon. (Mr. Gould here applied the strapping to a young adult according to Sayre's method.)

Now, Gentlemen, this is an example of a rule which I have not yet mentioned except incidentally—*Never use a splint to correct deformity.* (The strapping represents the splint in this case.) *First correct the deformity, then put on the splints;* and it is that point I wanted particularly to illustrate by this treatment of fractured clavicle. Get the arm drawn forwards—which means the shoulder forced backwards—and then put on your strapping and hold it firmly in position. Do not trust your strapping to draw the arm forwards, because it won't. If you have a broken limb to put up, first correct the displacement and then put on your apparatus.

We have here a patient, a nurse, with a *Colles' fracture*. She met with an accident, and broke her radius last Thursday. It was put up in what I may

call the ordinary way; and I saw her on Monday. The hand was then very much swollen. It was between two straight splints; and there was a good deal of pain. I then put on a splint which all of you may not know, namely, Carr's splint. If you ask her she will tell you how very much more comfortable she has been since she had this splint. The point that I want to mention is that Colles' fracture has two evil consequences—deformity (some people do not think much of that) and stiffness of the hand. Now do remember that the great object in the treatment of every fracture is to restore the function of the part. Surgeons may take great trouble fixing the two bits of the radius, and not think enough about the glueing of the tendons that is going on all the time, and as a result they turn out their patients with stiff hands. It would have been better for these patients never to have seen a doctor. I have seen more than one patient who has not been to a doctor at all for a Colles' fracture, and has come to me because of the deformity, who has had a much better result than if treated in the old-fashioned way, with a pistol splint, and I have silently congratulated them on their good fortune. Do remember it is the restoration of a useful hand you have to think about. Besides restoring the fragments to their position there is only one additional secret for restoring the function, namely, to put on an apparatus that leaves the fingers and thumb perfectly free to move. That is the merit of this splint of Carr's. It consists of a palmar splint with a thick radial border which comes under the shaft of the radius, and an oblique roll, which the patient grasps, comes into the palm of the hand and projects between the thumb and forefinger; and then a dorsal splint. These are held in place by a bandage at first, and later on by two straps and buckles. By this apparatus the radius is fixed while the thumb and fingers are perfectly free to move. The patient is told to move her thumb and fingers freely, and in this way you can make sure the tendons are not fixed. Prevention is vastly better than cure. You know how exceedingly painful it is to break down adhesions that have once formed, and how tedious such cases are; you will generally get a perfect result so far as the use of the limb is concerned by the treatment of which I have spoken. So far as the deformity is concerned you rarely get quite a perfect result.

Fracture of the fingers was the only other fracture I was going to mention, and I have only time just

to caution you against amputating fractured fingers unnecessarily. There is no class of injury which gives you such surprising results as compound fractures of the fingers. I could quote cases to you where the fracture has been into the joint, and so on, which recovered perfectly with freely movable fingers.

A CLINICAL LECTURE

ON

A CASE OF PYOSALPINX.

Delivered at the London Hospital, by

G. ERNEST HERMAN, M.B. Lond., F.R.C.P.,

Obstetric Physician and Lecturer on Obstetric Medicine to the Hospital.

GENTLEMEN,—I speak to you to-day about a case of disease of the Fallopian tube. The complaint which brought the patient to the hospital was a severe pain which she had had ever since her confinement. From *her* point of view that severe pain constitutes her illness. From *our* point of view when we came to examine the patient, we found a lump behind and on one side of the uterus, a lump which was fixed and very tender on pressure. I ask you to notice that it was a lump, and not mere induration; it was not that the layer of peritoneum covering the pelvis was thicker and harder than usual, but there was a distinct lump which, on bimanual examination, could be grasped between the internal and external hand. The patient had been in hospital for three months, during which she had rested in bed, and had other treatment under which the lump behind the uterus got much smaller; but it had not gone away and the pain had not decreased. After having been out of the hospital for about a fortnight, the patient came back saying that her pain was so bad she could not keep about. There was nothing that, from her point of view, was of moment about her case except that feature: severe persistent aching pain, with exacerbations of great severity—pain so bad as to prevent her from sleeping and from doing her ordinary household work. She was then again admitted into the hospital, and an operation was performed.

You may ask, Why was the operation done? Was it to relieve her pain? It was not done to

relieve her pain, but because it was believed that her pain was caused by disease which was incurable except by operation, and which might be fatal. You may further ask, Why was not the operation done before? Why keep the patient in hospital three months before operating? This was done because the diagnosis between the different conditions which may give rise to painful fixed lumps in the pelvis is so difficult that we cannot be certain of their nature except by watching the course of the case under treatment.

A tender fixed lump in the pelvis may be one of several conditions. In the first place, it may be simply a mass of bowels matted together by adhesions with some serous fluid in the spaces between these adhesions. That inflammation may have been set up by some cause which has ceased to exist. For instance, it may be the result of cold. It is doubtful whether the affections which we think due to cold are not really due to the entrance of some micro-organism. That may be so; but if so, it is a micro-organism which the body has the power of destroying, because these affections for which we can find no other cause than cold get quite well. In a lump of this kind, the fluid in the course of time gets absorbed, the adhesions become looser, and the lump ceases to be. There is only one way of being sure that a painful fixed lump in the pelvis is not a lump of this kind, and that is by watching its course and seeing whether it will not go away under treatment. If it is simply an inflammatory affection of the peritoneum, and nothing else, the state of things present will, under treatment, in the course of time more and more approximate to the condition of health. That is the feature common to all inflammatory diseases, as opposed to new growths. New growths have no tendency whatever to go away, whereas inflammatory diseases when once the cause is removed tend more and more to approach the condition of health. We often see lumps like this in the course of time altogether disappear, or become so small and ill-defined that they cannot be recognized as lumps. This lump during the patient's stay in the hospital diminished considerably in size. When she came in it nearly filled the pelvis, and pushed the uterus forward. At the time of operation it was much smaller, and confined to the space adjoining one corner of the uterus. Such painful fixed lumps in the pelvis, then, may simply consist of bowels matted together by adhesions, with a little fluid

between the adhesions, but without any permanent disease of the Fallopian tubes or ovaries.

Another possibility is that such a lump may contain in it a focus of suppuration. If so, it will never get well as long as the cause of suppuration is present. The suppuration may be in the Fallopian tube or in the ovary. The commonest seat of suppuration is the tube. Suppurated tubes are found about twice as often as suppurated ovaries; and the suppurated ovary is generally cystic—there is a suppurated ovarian cyst. If, when we have a tender fixed lump in the pelvis, we could diagnose it at once as due to a focus of suppuration in its centre, we should not delay, but operate at once; but when we cannot be certain, the only way to make sure is by watching the course of the disease. We may suspect it although we cannot be certain of it.

I have said that if we were sure there was a suppurated tube or ovary we should operate at once. But is it quite certain that suppurated tubes never get well? I have little doubt that they sometimes do get practically well, that with time the inflammation ceases, the micro-organisms which caused the disease die, and the pus undergoes changes, becoming caseated or even calcified, so that the patient remains with a tube which shows the changes of past disease but not of active inflammation. I am sure from clinical observation that a patient may go on for years with a fixed lump in the pelvis without the lump causing any kind of suffering. But we cannot in the least count upon that: it is one course which a suppurated tube may take, but we never can be sure that it will take that course. If a patient has suppuration in the Fallopian tube she is liable, in the first place, to recurrent attacks of local peritonitis around the tube. In the next place she may get fatal general peritonitis starting from the suppurated tube. Statistics show that in patients with suppurated tubes these lead to death in about one-fourth of the cases; so that although it is possible that a suppurated tube may get well or pass into a chronic state in which it neither causes suffering nor endangers life, yet such an event cannot be counted on.

A case such as this, in which the severe pain distinctly followed delivery and persisted afterwards, is not likely to be anything else than one of these three conditions. There are, however, other lumps in the pelvis which, so far as physical

signs are concerned, cannot be distinguished from those of which I have spoken. Among these we have, for instance, hydrosalpinx, that is, distension of a Fallopian tube with watery fluid. This is a chronic condition which is seldom associated with acute peritonitis, but is generally mistaken for an ovarian cyst, from which it is difficult to distinguish it. A hydrosalpinx may be formed out of a pyosalpinx. When the inflammation ceases in a tube which originally contained pus, the pus may caseate, the fat globules becoming aggregated together into a caseous mass, and the fluid remaining. In the same way hydrosalpinx may result from the effusion of blood into the tube, the blood coagulating, the coagulum becoming more and more decolorized, and the serum remaining. Hydrosalpinx produced in either of these ways is not an acute but a chronic condition. It rarely causes such severe pain as that from which this patient suffered, so that it was not thought likely that this case was one of hydrosalpinx. A tumour formed by hæmatosalpinx cannot so far as its physical signs are concerned, be distinguished from one formed by pyosalpinx; but it does not commonly come on after delivery. Its common cause is tubal gestation, and that is not likely to be the case here. Hæmatosalpinx may get well by absorption of the blood, and effusion of blood into the peritoneum may also get well without operation; so that if we were quite certain that in our case one of these two conditions was present we should not be urgent about operating. Such a lump might also be an ovarian tumour, or a fibroid, fixed by peritoneal adhesions. But unless there were in the tumour changes keeping up inflammation, there would not be such severe persistent pain as in this case.

To return to our patient. She had a lump in the pelvis. From the fact that while the patient was for three months under the most favourable conditions the lump had not gone and the pain had not ceased we inferred that it was not due simply to perimetritis, but that there was more than this. As the lump has not been absorbed, we concluded that it was not an effusion of blood; that probably there was pelvic suppuration. But there was nothing in the history of the case to enable us to say whether the suppuration was in the tube or the ovary.

I have stated that this patient had severe, persistent pain, but have not gone into details as to the precise seat of the pain, because that was not

important. The pain was chiefly in the lower part of the abdomen, more particularly on the left side. After she got up she noticed pain in her back passage, and had bearing down pain. Her pain was extremely aggravated by defæcation. I was told that it was quite painful to see her when using the bed-pan. This pain in defæcation is common in such cases because the presence of this inflammatory lump close to the rectum causes congestion of the mucous membrane of the rectum, which becomes tender; the bearing down accompanying defæcation causes movement of and pulling on, the inflamed parts; the passage of fæces over the inflamed part irritates it, and all this causes pain.

There is generally uterine hæmorrhage in these cases of inflammation of the tubes. Although there is usually increased menstruation—menstruation being irregular and increased in quantity and frequency, that is not of importance in diagnosis.

As to the method of diagnosis. These lumps formed by suppurated tubes or ovaries are very painful and tender, so that it is often difficult to examine the patient, and if the lump is small, difficult to be sure there is a lump at all. In such cases the best way of making a diagnosis is to examine the patient under an anæsthetic. Thus you get a completeness of exploration not obtainable in any other way. To ascertain the condition of the uterine appendages on each side, use for vaginal examination the hand corresponding to the side you want to explore. To examine the ovary and tube on the right side, stand on the right side of the patient, put the first two fingers of the right hand into the vagina and the left hand on the abdomen. To examine the structures on the left side, put the two fingers of the left hand into the vagina and the right hand on the abdomen. If this does not enable you to make up your mind as to the condition of the uterine appendages, a good plan is to put two fingers into the rectum. In this way you get higher up behind the uterine appendages than you can by the vagina, and you can explore them thoroughly. You can only do that under an anæsthetic.

There are one or two points about the physical signs of these lumps that I may refer to. You will find it stated that the swellings formed by diseased tubes are sausage-shaped or pear-shaped. It is true that the distended tube when taken out of the body is often of one of these shapes. As you meet with them in the body, however, they are so

closely united to other parts by these adhesions, that it is exceptional to be able to make out during life the shape of the distended tube. This fact, therefore, that the tubes when distended become sausage-shaped or pear-shaped, does not help in diagnosis. Neither does the closeness or otherwise of their connection with the uterus help you much, because, although the swelling formed by a dilated tube is mainly in the outer part of the tube, and, therefore, you may think ought not to be continuous with the uterus, yet the parts are commonly so blended together by adhesions, that you cannot trace any distinction between the tumour and the uterus, so that in most cases you do not get much further on bimanual examination than this, that there is a swelling which is close to the uterus.

I have said that in this case the operation was done not because of the pain, but because it was believed that the pain indicated the existence of a serious disease which it was necessary to cure and which could not be cured in any other way. You may ask, Why not operate if a patient has severe pain? Why not remove the painful parts whether the disease be serious or not? The reason why it is not desirable to remove the uterine appendages merely for pain is because operations of this kind simply for the relief of pain are generally failures. Two things go to produce pain. One is local disease, the other the state of the patient's nervous system. In some patients there is great local disease with but little pain; while in others who complain of great pain there is but little local disease. If a patient complains of pain in the pelvis and there is no local disease, it is certain that removal of organs from the pelvis will not cure the patient; while if the disease in the pelvis is only slight the chances are that after an operation for removing this slight disease, the patient will not be relieved of the pain. If, on the other hand, the patient has serious disease in the pelvis, even if you do not completely cure the patient of her pain, you do good by removing the serious disease that might prove fatal.

As to treatment.—When you have a case with a painful, tender fixed lump in the pelvis which you suppose to be inflammatory, but are not sure whether or not the inflammation is due to disease which will not get well without operation, you must keep the patient absolutely in bed and watch the progress of the case. Treatment of these cases is useless unless you secure rest in bed; and that is not easily done, especially in patients of the poorer

classes who cannot get rest at home so thoroughly as they do at the hospital; and even in the patients more comfortably situated it is sometimes not easy to secure.

To prevent the irritation of the bowel, which is so troublesome in these cases, it is desirable to give laxatives so as to keep the motions somewhat loose.

Only one remedy has a direct effect on inflammation within the pelvis. No drug will arrest the course of inflammation within the pelvis, but there is a remedy that will modify its course. You can see it lessen inflammation in the eye. If you have a patient with inflammation in the conjunctiva and you put a blister behind the ear, you can see the effect. In the case of inflammation in the pelvis the same beneficial result follows counter-irritation. Iodine, blisters or mustard poultices applied to the abdomen generally relieve the pelvic pain.

There are other kinds of treatment advocated, and of some of these I may speak. Electricity has lately been much spoken about. I do not think that electricity does any good in these cases. Neither faradic electricity nor galvanic electricity put through a lump like this do I believe to be of the slightest benefit either in relieving pain or affecting the morbid condition. Galvano-puncture has been advocated, but it is not safe. If you interfere it is much better to do so in other ways. Massage is sometimes recommended in these cases of pelvic pain. If you have a lump which is formed of a suppurated tube or ovarian cyst there is danger in abdominal massage, since manipulation of the abdomen may burst the abscess, and fatal peritonitis ensue. I have nothing to say against massage of the limbs. No doubt it is beneficial to the general health; but it will do nothing towards removing the pelvic disease.

One object obtained by operation which is sometimes recommended as in itself a reason for operation, is the separation of adhesions. I am unable to see how, if adhesions are separated by operation, the re-formation of adhesions is prevented. There are some few cases in which it is said that a good deal of pain has been caused by adhesions of the omentum to the pelvis pulling on the stomach and intestine; and I quite believe that good may be done by separating adhesions of this kind; but I know of no way in which the presence of these adhesions can be diagnosed, and therefore

I do not at present recommend operations for that purpose.

Supposing we have a case of a fixed tender lump in the pelvis, which we have treated for, say two or three months, without effect. I think that we ought not to assume that treatment will be in vain until at least two months have elapsed, for I have watched cases which only after that time have begun to improve, and yet have finally left the hospital without pain and without a lump. If after two months treatment the pain and the lump are undiminished, do not wait much longer. If the pain is still so bad as to interfere with the patient's capacity for fulfilling her ordinary duties, the only way is to give surgical relief.

There are two ways in which surgical relief can be given. One is by the vagina, the other by the abdomen. To attack the disease by the vagina is only desirable if the lump we are dealing with is fixed low down behind the uterus, in which case the attempt to give relief by this method can do little harm. If it is not firmly fixed in Douglas's pouch, it is better to approach it by the abdomen; and there are good reasons for preferring operation by the abdomen, even when the tumour is fixed behind the uterus. For instance, you may have one suppurated cyst fixed behind the uterus, while there are other cysts which make removal of the whole ovary desirable; by attacking the tumour through the vagina you will only empty the one suppurated cyst. The best way of performing the operation through the vagina is to make a free opening in the vagina behind the uterus with the scissors. When you have got into the pus-containing cavity, put in two fingers and freely enlarge the opening to it. Put your other hand on the abdomen and get the fingers of the internal hand, if possible, right to the end of whatever lump you feel, breaking down whatever partitions may exist within the lump so as to thoroughly open up all the cavities it contains. Stop hæmorrhage if necessary by plugging with Iodoform gauze, or by stuffing with a clean dry sponge liberally dusted with Iodoform, and having a string tied round it by which it can be removed. I have treated cases in this way, and with satisfactory result. But the majority of cases cannot be treated in this way. In the case of which we have been speaking, the lump was high up and not within reach by the vagina. It was attached to the upper and left angle of the uterus. Another stronger reason for the abdominal operation, and against the vaginal, is

that of suppurated lumps within the pelvis, one-third are suppurated ovarian cysts. Now pyosalpinx can be cured by vaginal incision, but suppurated ovarian cysts cannot always be so cured.

Operation was performed in this case. When the abdomen was opened the intestines were found matted together in and above the pelvis. A hard lump was felt in the left half of the pelvis. The adhesions around the lump being broken down, the tumour was got out. A little pus came from it during removal, showing that it was a pus-containing cavity. I showed it at the last lecture, and the greater part of it was found to consist of the outer part of the Fallopian tube, which was dilated, containing pus, and greatly thickened by fibrous tissue. The rest of the lump consisted of the inner part of the Fallopian tube, which was convoluted, thickened, but not dilated.

It is not my purpose in the present lecture to speak of the details of these operations. I may just mention that the pelvic contents were matted together by adhesions; and that on the other side of the pelvis I could not identify the Fallopian tube or ovary at all, these being closely adherent to the bowels. There being no lump on that right side like that on the left I left them undisturbed.

The result has been that the patient is now getting up and is free from pain. It is too soon to speak as to the ultimate result. The operation was done a month ago. You may ask, What is the usual result of such cases? Using the word "cure" to indicate the attainment of permanent good health, they are not all cured. This is on account of the difficulty of diagnosis. No doubt when diagnosis becomes more accurate we shall be able to identify beforehand the cases which are curable, and those which are not. Different estimates vary. According to some about half or a little over a half get cured, according to others about four-fifths. The estimates of different operators vary between these proportions.

Supposing the patient is not cured what evil results may ensue? In the first place the operation may prove fatal. The mortality of this operation varies like that of all operations according to the operator's skill and experience; but taking the results obtained by those who have performed the operation many times the mortality seems to be between 5 and 10 per cent. The experience and the skill of the operator go together, because these operations cannot be practised on the dead

subject. There is only one way of learning how to do them, and that is, by first seeing them done and then doing them on the living subject. Therefore, the larger the operator's experience the better the results he obtains.

Persistence of the pelvic pain may be another undesirable result. This is the case in some instances, and the more the operation is done in cases in which the pelvic organs are very little diseased the larger will be the proportion of cases in which the pain persists.

In these cases the diseased parts are generally adherent to the bowels, and even the most experienced operator may tear holes in the alimentary canal, the result being a fæcal fistula. In some cases these fistulæ persist a long time, even for months and sometimes years. All the cases I have seen have healed up in the course of a few days, but in the experience of very distinguished operators there have been fæcal fistulæ lasting for years; and one must take that as one of the possibilities attending operations of this kind.

Ventral hernia is another, though trifling result. Where the abdominal wound was made there is a weak spot; and through it a hernia may protrude. That is not important, however, as such herniæ are easily cured.

Taking the mortality of these cases at from 5 to 10 per cent., while you remember that of suppurated tubes about a quarter cause death, you will see that as a means of preventing a fatal result these operations are justified. When you recollect also that at least the majority of these operations cure or relieve the patient's suffering, you will acknowledge the operation is abundantly justified. The only ground for postponing the operation is the uncertainty of diagnosis, since we can only suspect the presence of suppurated tube or ovary. The failure of other treatment indicates the necessity of operating. Operation might be resorted to more frequently; but if so, we should be certain in some cases to perform it where perfect recovery would take place without it.

THERAPEUTICAL NOTE.

Peripheral Neuritis, Alcoholic.—Dr. v. Boltenstern relates a very severe case of this disease, in which the paralysis of all four limbs was nearly absolute, where the hypodermic use of Strychnine

proved of most signal service. He uses a 1 per cent. solution of Nitrate of Strychnine, and begins with a dose of \mathcal{M}_j , increasing it daily by \mathcal{M}_j up to \mathcal{M}_x , and then an intermission of a few days follows, and then \mathcal{M}_x are used every day for nine days, a second intermission, and so on in alternation. He reports the case as cured in four weeks from the commencement of the injections. Warm baths with cold douches also assisted in the cure.

Therap. Monats., 8, 1893.

REVIEWS.

Tumours: Innocent and Malignant. By J. BLAND SUTTON, F.R.C.S. (Cassell & Co.)

Published at 21s.

To a mind trained on mathematical and natural scientific lines nothing is more refreshing than to find scientific principles brought to bear on professional writing. Mr. Sutton has succeeded in doing this in a most admirable manner in his book on tumours. Starting very much on the lines first laid down by Virchow, the author commences the description of each group with a beautifully simple and scientifically correct classification of its individual members. Nomenclature of tumours is thus reduced to its simplest form, and the pages of the book are not burdened with the weight of lists of ungainly names.

In the introductory chapter Mr. Sutton specially excludes the infective granulomata; their exclusion is, however, to be little regretted by reason of the fuller discussion of other tumour formations.

The first seventeen chapters are devoted to the macroscopical clinical features of Group I., known as connective tissue tumours, of which he makes twelve genera, corresponding to the adult tissue simulated by the neoplasm: the microscopical characters are described only so far as clinical needs are concerned, the book being intended as a practical guide for surgeons and not as a microscopist's vade-mecum. All the genera are discussed excellently, but the description of the odontomata requires a special word of praise; the author draws attention to the fact that an adult tooth is really a compound organ, and each of the elements in it is capable of hyperplasia with the

formation of a tumour; consequently, there are no less than seven species in this genus—one starting from the enamel, four from the follicle, one from the papilla, and one from the whole germ. No less than seven chapters are devoted to sarcomata, divided into six species according to the prevailing shape and other characters of the cells; these chapters are exhaustive of the principles of sarcomatous growths, and every case occurring in practice will find its explanation here. Rhabdomyomata are referred to as a species of sarcoma, and neuromata as tumours of the *sheaths* of nerves, and ample justification for both positions is found in the facts brought forward.

Group II., tumours of epithelial origin, occupies the succeeding eleven chapters; these Mr. Sutton divides into genera and species, and simplifies in a most charming manner the difficulties which have so long encompassed any general description and study of these neoplasms. The genera are—papillomata with four species, epitheliomata one species, adenomata and carcinomata each with fourteen species, corresponding to the organ from which they spring.

Group III., or dermoid tumours and teratomata, occupies twelve chapters; dermoids are classified in four genera with true scientific distinctions, and order comes where chaos reigned; the genera are—sequestration-dermoids arising from a portion of misplaced surface epithelium, tubulo-dermoids from permanence of foetal ducts, ovarian dermoids in connection with ovarian tissue, and, lastly, dermoid patches, moles, etc.

Group IV., cysts and pseudo-cysts, occupies ten chapters, and includes tubulo-cysts, neural cysts, hydatids, etc. The book concludes with two very interesting chapters on the causation of tumours in general, and their distribution amongst the members of the whole animal kingdom.

We have only briefly drawn attention to the excellence of the pathological side of this work, but it is from a surgical aspect that it will be mostly prized. The sections in each group dealing with treatment are admirable, and will prove a great boon to practitioners living at a distance from consultative centres. Brief, but most excellent and clear, directions are given for the necessity or expediency of operation; the methods of operating are thoroughly well described; in a few cases the absolute futility of any operation is clearly shown.

We cannot conclude without praising the ex-

cellence of the illustrations, of which there are 250 beautiful cuts and nine full-page plates, some of which are coloured; they all, without exception, reflect the greatest credit on the draughtsman and engraver. Throughout this book Mr. Sutton has given the most careful recognition to the work of others, and no report of cases appears without a full reference. A most useful and correct index closes a work, the perusal of which has given us very great pleasure.

WE have received from Messrs. Danielson & Co., two specimens of diagrams of the brain, engraved on slates, 12 by 9 in., price 12s. 6d. each, or £6 for a set of 10. When we say that they have been inspected by Victor Horsley, we cannot say more for their accuracy, which struck us as remarkable. They will form excellent additions to the P.M. room for recording situations of lesions, and also to the dissecting room for teaching purposes. Being engraved on slate they are, with fair usage, indestructible, and an infinite number of paper rubbings can be taken from them for use in note-books. The idea is an excellent one, and we hope and think the slates will soon find their way into every hospital for use in wards, post-mortem, and dissecting rooms.

THE 5th year of the London Post-Graduate Course commenced on January 15th, 1894. The course as usual consists of Clinical Lectures and Demonstrations, given at the following hospitals by members of their respective staffs:—

HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST, BROMPTON. HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET. NATIONAL HOSPITAL FOR THE PARALYSED AND THE EPILEPTIC, QUEEN SQUARE. ROYAL LONDON OPHTHALMIC HOSPITAL, MOORFIELDS. HOSPITAL FOR DISEASES OF THE SKIN, BLACKFRIARS. BETHLEM ROYAL HOSPITAL FOR LUNATICS. LONDON THROAT HOSPITAL, 204, GREAT PORTLAND STREET, W. BACTERIOLOGICAL LABORATORY, KING'S COLLEGE.

Further, Clinical Lectures on appropriate cases will be delivered at the Sick Asylum, Cleveland Street.

Daily classes will be held, during what are termed Vacation Courses, in Bacteriology, Pathology, Hygiene, and other subjects, during the fortnight preceding the commencement of the Summer Term and of the Winter Term, and during the first fortnight in December. Subscribers have the option of joining any one or more classes. We cordially wish this valuable course a success equal to its high merits.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 24, 1894.

ORIGINAL ARTICLE.

DILATATION OF THE SPHINCTER ANI.

By T. OLIFFORD ALLBUTT, F.R.S.,
Regius Professor of Medicine, Cambridge University.

IN the bright and useful lecture on hæmorrhoids which Mr. Treves has contributed to the many of such lectures which have appeared in the "Clinical Journal," Mr. Treves makes a statement which I will venture not perhaps to traverse but to enlarge. I am no surgeon, and I make the venture with some trepidation.

The statement is taken from the fourth to the eighth line of the first column of page 121 in the third volume (No. 8, Dec. 20, 1893), and I quote it as follows, premising that the context is concerned with the treatment of hæmorrhoids.

"(3) *Dilatation of the sphincter (ani) with bougies* is not of very much value. It has been assumed to act by the rest that is necessary both before and after the bougie is passed. A large bougie is used. How it acts it is difficult to say."

Now this statement I venture to enlarge, and this by removing in the first instance the words "with bougies" in the first line, and of course the subsequent references to bougies which follow. The statement would then run thus:

Dilatation of the sphincter is not of very much value. It has been assumed to act by the rest that is necessary, both before and after the operation. How it acts it is difficult to say.

Again I will venture upon a defalcation. I will omit all that part of the paragraph which concerns itself with the explanation of the mode of relief, if any. These latter words I will omit for two reasons; first, because I have, for a physician, a large experience of cases of rectal troubles, treated either by rest, or by dilatation of the sphincter ani, or again by combinations of the two; and I have satisfied myself by such experience that rest is not directly concerned in the relief often attained by dilatation. Patients suffering from disorders of the rectum may be confined to bed for two or three

days or more with but little permanent relief or none; on the other hand, patients may be submitted to dilatation in the surgeon's chambers, and return home within a few hours without thereby forfeiting the benefits of this operation.

To return to the liberties I have taken with Mr. Treves' text: I have removed the words "with bougies" because I believe that this method, however useful for the gradual enlargement of a rectum reduced by cicatricial contraction, is an inefficient method for dilating a contracted sphincter. The only efficient way of dilating such a sphincter is by the insertion of the two thumbs of the operator into the orifice, and the forcible separation of them until the muscle is felt to creak and give under the strain. The bougie conveys no sense to the operator of this degree being reached, nor, on the other hand, of any undue laceration of that part, but the practised touch of the surgeon recognizes readily the degree of stretching which is efficacious on the one hand and safe on the other.

I may add that in many cases this degree is no small one, and, in the case of a toughened adult sphincter, may need something like the full strength of the operator (placed, as he is, at some disadvantage) to attain it. Dilatation by bougie often falls far short of this efficient degree, and therefore needs repetition, even frequent repetition. One dilatation by the thumbs should be final; it can rarely be proposed without an anæsthetic, but is, even thus, more tolerable than the recurrent annoyance and pain of bougie treatment. If any incontinence follow, it never lasts for more than a day or two. So far, then, in respect of method.

As regards purpose, I may point out that tight sphincters (and it is needless to say that lax sphincters do not require such treatment) are of two kinds: (a) Sphincters which are normal in structure, but abnormal in function; (b) Sphincters which are abnormal both in function and structure. To take the case (a) first: in such a case a sphincter, not obviously abnormal in structure, may be found in a state of tight contraction, which contraction may be due to irritation, perhaps acting upon it directly, more probably by way of reflex action. In case (b) we find a sphincter

either simply hypertrophied, presumably by prolonged overaction, or hardened into a comparatively rigid, quasi-cartilaginous ring. Whether this latter state be a consequence of simple hypertrophy I do not know.

There are speculative persons who say that whatever "Nature" does "she" does with a curative or conservative purpose; I should no more apply an assumption of this beneficent tendency to a given case than I should apply the converse. Were there not a large margin of safety in "Nature's" system our lives would be worth little; when the consequences of a perturbation fall within such a margin, "Nature" is seen to be conservative; when they fall outside it we die. In the case of recovery it does not follow that each several event of this series was conservative, it means only that the sum of them was conservative; each event must be judged as it severally contributed to life or death.

Now it very commonly happens that in a series which tends on the whole towards recovery, events and processes are contained which are mischievous: perhaps no morbid series, however favourable its tendency to recovery, can be found in which there are no events or processes which counteract and delay this generally favourable course. Were it otherwise, our place as medical artists would be gone. Indeed, we not infrequently find something like an equation of counteracting processes so that the patient remains in an approximately stationary state, the forces tending to recovery being neutralized by opposite forces. Thus so vicious a circle may be established, that to use a current saying a patient "must get worse before he can get better," that is, if recovery is to take place, the tension between these opposing forces must be released—the "deadlock" must be overcome—even if some considerable oscillation in the direction of danger have to be reckoned with.

Such conditions I believe to exist in the very common cases of piles, fissures and the like, in the rectum: a spasm of the sphincter, by reflex action or otherwise, is set up; it is a blind and usually mischievous event, and one which counteracts the processes of recovery. The vicious circle must be broken, and it is often broken by forcible dilatation of the sphincter, so that the patient recovers promptly. I do not pretend to explain either the harm of the spasm or the benefit of its abolition, but I am bold to declare the beneficial results of the interference.

A quarter of a century ago, this treatment was recommended to me by Mr. Teale; not a year has passed since that time without many illustrations of its value in cases thus operated upon for me by Mr. Teale, Mr. Jessop, and other able surgeons who are witnesses of its success. Many are the patients who come to the physician for constitutional disorders due to or aggravated by piles; many of these have been treated by one direct operation or another without permanent cure, and in these a forcible dilatation will often, I may say, will generally, effect a cure of the piles by some indirect process. *Experto crede, inexperto exploratum sit.*

In this article I have not contemplated piles only, but also certain irritations of the rectum due to other causes of a curable kind. Of such disorders I will give a striking example. Six or seven years ago a lady of middle age or more, came to me saying that she believed her call upon me would be bootless: she had suffered for two or three years from a continual weary aching about the pelvis and beyond it, the origin of which no one could discover. The pain had not only embittered her own life but had so altered her disposition that her children had grown weary of her fretfulness, and an unmarried daughter who accompanied her gave me at last to understand plainly that she was growing weary of her mother's complaints, and was coming to the end of her own patience. She had the less hesitation in saying this as her mother had been under the care of one able specialist and another, who all assured her that there was nothing the matter with her. She was the head of a large and influential family, and covert proposals had been made to remove her from home, and place her under some modified kind of "mental treatment." Before falling in with these counsels I examined her minutely—her nervous system, her uterus, and the rest of it. Finally, I examined the rectum, as she complained of constipation: I found the gut was *hot*, but it presented no other abnormality to the finger except a densely hard tight sphincter: the sphincter Mr. Teale stretched bimanually, as above described; recovery was speedy and complete.

I will only add that many cases of obstinate constipation, especially in women, are due to tightness of the sphincter ani, or, to speak more carefully, are relieved by forcible bimanual dilatation of it.

A CLINICAL LECTURE ON THE TREATMENT AND MANAGEMENT OF PULMONARY TUBERCULOSIS.

Delivered at University College Hospital, by

G. V. POORE, M.D., F.R.C.P.

Physician to the Hospital.

BEFORE coming to details of treatment and management, and in order that any recommendations I may make to that end may appear reasonable and justifiable, it is necessary to give a comprehensive glance at certain points in the ætiology and pathology of tuberculosis, concerning which there is now a very general agreement.

The labours of many statisticians during the past half century have shown conclusively that the death-rate from phthisis bears a very general direct proportion to the degree of concentration of population. It is essentially a disease of cities, and amongst dwellers in cities and large towns, its effects, especially, those who are engaged in sedentary indoor employments such as clerks, shop-assistants, compositors. Where many persons are congregated under a common roof, with insufficient cubic space, phthisis is prone to be very rife, especially if the other hygienic conditions are defective. We accordingly find that the death-rate from this cause in barracks and prisons formerly reached a very high figure. Now, however, that greater attention is paid to cubic space and ventilation the death-rate from phthisis among soldiers and prisoners is very much lessened. It is important to bear in mind that in comparison with overcrowding, and especially overcrowding *indoors* and under a common roof, all the other ætiological factors of phthisis hold a very subordinate position. It is a disease of all climates, and its occurrence does not seem to be much influenced by latitude. In warm climates where city populations bask *out of doors* in the sunshine there is generally speaking less phthisis than in cities which have a rough and variable climate like Vienna, a city whose inhabitants suffer more from phthisis than those of any other city in the world. In Vienna the population is largely devoted to indoor industries, and the inhabitants live in large tenement houses which are warmed by stoves. The variations in temperature are often sudden and extreme, and few who have

been there in winter will forget the cutting severity of the wind. There are certain islands in Europe which are said to enjoy an immunity from phthisis which is more or less complete, and it is interesting to note that these islands—Iceland and the Faroe Islands—have cold and severe climates. It is also interesting to note that among European cities the northern cities—Christiania, Copenhagen, Stockholm, Königsberg—have a slightly lower death-rate from phthisis than some of the cities lying in warmer latitudes. Other things being equal it would seem that elevation above the sea-level lessens the tendency to phthisis. The plains are, as a rule, more crowded than the mountain-tops, and there can be no doubt that the immunity of mountaineers is due, not only to their well-developed chests and athletic lives, but also to the sparse population of the regions which they inhabit.

Having arrived at the conclusion that the main factor in the causation of tuberculosis is overcrowding, the great discovery of Koch comes, as it were, to give additional point to our conclusion.

Before Koch's discovery medical opinion was becoming more and more firm as to the infective nature of pulmonary tuberculosis, and the discovery of a *contagium vivum* in the form of the tubercle bacillus converts a theory into a fact, and gives us a solid basis both for treatment and prevention.

While one must not forget the possibility of the conveyance of tuberculosis by tuberculous meat or milk (uncooked), we shall all readily admit that the usual channel of conveyance is the air, and that tuberculosis, although a chronic trouble with a doubtful incubative period, must, for practical purposes, be classed with *air-borne* contagia, such as diphtheria, and the other infective fevers which are conveyed through the air. Although it is possible that the air may be infected by the coughing and sneezing of tuberculous patients, still, the most probable source of infection seems to be from tuberculous sputa, which, becoming dried, are raised in the form of dust.

It stands to reason that the danger of being infected in this way is much greater indoors than out of doors. Tuberculous sputa falling out of doors probably quickly succumb to the saprophytic organisms which abound everywhere. When, on the other hand, they fall indoors, they dry up, and not being kept moist, putrefaction is delayed, they do not succumb to saprophytes, but, being persistent spore-bearing organisms, they remain

ready to commence growing so soon as they fall upon a congenial soil. A volume of air which is enclosed in a room cannot, in spite of all our efforts, be renewed with anything approaching the completeness and frequency that a similar volume is renewed when not so enclosed. If we assume that a given volume of air contains a certain number of infective particles, it follows that such particles are the more likely to infect, the greater the difficulty of renewing the air. Hence it follows that our danger of infection from all air-borne contagia is much greater indoors than out of doors. Given the number of infective particles in any volume of air, the danger of infection might be stated mathematically. There seems however, good reason to believe that overcrowding in rooms and houses intensifies the danger of infection from air-borne contagia, apart from mere mathematical considerations. It is evident that if our danger of infection is due to the particles of dried sputum raised in dust, that the stirring the air and raising the dust, which is much greater in a crowded room than an empty one, is, in itself, a source of danger. Again, it appears probable that air which is heavy with carbonic acid and watery vapour, has a greater power of carrying floating particles than air which is pure and dry, and thus we are able to adduce two physical reasons which may account for the danger of overcrowding, apart from the merely mathematical considerations based upon a knowledge of the cubic contents of the room, and the number of sources of infection. It is probable that there are other causes besides physical ones which enhance the danger of overcrowding, and it may well be that the difficulties experienced in the proper aeration of the blood may increase the liability of the infective particles to "take root" in the individual who inhales them. All these considerations show how important it is that sputa should be received into vessels, and be systematically burnt or chemically disinfected.

It must never be forgotten that the liability of the infective particle to "take root" and grow varies with the individual. Some are very susceptible to phthisis, others very insusceptible. Tuberculosis is so common that one would suppose that in a great city like London there must be large numbers of tubercle bacilli floating in the air, and yet comparatively few of us become definitely infected. It must be supposed that a large number of those who get infected, practically recover without suffering seriously, *i.e.*, they throw off

the infection. We know how common it is in the post-mortem room to find lungs adherent at the apex, or calcified or caseous glands in subjects who have never been regarded as phthisical, and we may justly conclude that a fair proportion of these appearances are the records of a successful fight with the bacillus tuberculosis.

It is difficult to say precisely what it is that causes the excessive vulnerability to phthisis in certain individuals, and why the bodies of some should prove a more congenial soil for the growth of tubercle bacilli than the bodies of others. Few of us doubt that a tendency to tuberculosis is distinctly heritable, and we know that the families in which such liability exists, contain a large number of individuals who are feeble, "lax-fibred," prone to anæmia, and very liable to catarrh. Not infrequently they have a characteristic figure, with a long narrow chest, and it may well be that with chests of such a shape the expulsions of bacilli which may gain access to the lung is no easy matter.

Now, our main subject, the prevention and cure of pulmonary tuberculosis may, perhaps, be best handled by considering what we can do to protect persons having a liability to phthisis from their probable fate.

In the first place, we must seek by fresh air and exercise to "develop the chest," and see that the expansion and contraction of the lungs are both improved by exercise. Walking, running, climbing or anything which calls for full inspirations will do this. Lagrange has pointed out that during exercise our need of deep inspirations is largely in proportion to the muscular mass which we put in action, and he, therefore, holds that the exercise of the legs in running is the best form of exercise to develop the chest. There are some who hold, as I hinted just now, that the immunity or comparative immunity from phthisis enjoyed by dwellers in mountainous districts, is due not so much to peculiarities of climate as to the well-developed chest of the mountaineers, whose deep and forcible respiratory movements give but a small chance for the permanent lodgment of bacilli.

While endeavouring to "develop the chest," we must take care that it is not overdone; for if, by excessive exercise, we allow the subject to get "blown" we may induce emphysema, which may give us enormous girth of chest with very feeble contractile power in the lung. Subjects who are prone to phthisis should carefully guard against

overtraining and exercises which induce breathlessness. The exercise should be carefully graduated, and the amount slowly and cautiously increased.

Such persons should be careful to avoid, if possible, "catching cold." This is a difficult matter, for the proneness to catarrh probably constitutes one of the chief causes of the liability to tuberculosis. Catarrhal secretions from mucous membranes obviously form a nidus for the growth and increase of bacilli; and there can be no doubt that a most important part in the prophylaxis of consumption in all its forms, is to keep the mucous membranes clean and free from catarrh. To this end there must be strict attention to diet, rigid temperance in the use of alcohol, and the avoidance of all sources of mechanical and chemical irritation, such as dusty atmospheres, and (especially, if there be a liability to laryngeal catarrh) tobacco smoking in all forms, inclusive of cigarette smoking.

Directly a patient gets a chronic cough, whether it be laryngeal, pharyngeal, or bronchial, there can be no doubt that his liability to become infected with tuberculosis is appreciably increased. The caution, therefore, to *maintain clean mucous membranes* is most important.

Persons who have a family tendency towards pulmonary tuberculosis should, if possible, live in the pure air of the country, and should select a profession or occupation which involves as little confinement indoors as possible. So long as they be free from actual lung disease, and provided they be warmly clothed and have a sufficiency of good wholesome simple food, I do not think it is necessary to "coddle" them too much. I am rather an advocate for sending them to tolerably bracing places, and rather prefer the east or south-east coasts of this country to the more soft and warm climates of the south and south-west. The life of a farmer or a sailor is the ideal life for these persons. I have seen, I think, some few lives saved by being taken from a close office and sent to work in the fields; and I can recall one case, the son of a mother who was the subject of pronounced tuberculosis, in whom the enlarged and shotty glands completely disappeared during two years' schooling at Ramsgate, and who became quite strong when he emigrated to Manitoba, where the extreme severity of the Canadian winter produced no ill effect.

If circumstances render it impossible to select a

completely outdoor life, we must do the best we can. Only the other day I saw the son of a small builder, who had never shown any manifestations of phthisis until he was moved from the carpenter's shop to the office, whither he had been attracted by the mistaken notion that the desk is a nobler field for energy than the bench of the handicraftsman. By my advice he left the office to enjoy the muscular exercise and the comparatively pure air of the carpenter's shop.

These delicate subjects should carefully avoid all overcrowded places, and especially in the evening, when the danger of catching cold on changing the stuffy atmosphere of the theatre or meeting-room for the chilly night air is very great indeed.

Now, when these persons develop the first signs of phthisis, we commonly send them away to some health resort, and it is well that you should thoroughly understand the object with which they are thus sent into temporary exile. The main object is that they should breathe as much pure air as possible.

Now, although cold cannot be regarded as in any sense a cause of tuberculosis, it is nevertheless a great source of discomfort and danger to those who, having become tubercular, are the subjects of that chronic pneumonia which is caused by the growth of bacilli in the lung, and which we usually speak of as phthisis. Persons with chronic lung disease are as a rule very intolerant of cold, and any undue chilling of the surface of the body is apt to produce a congestion of the lung, with an extension of bronchitic and pneumonic troubles. The Registrar-General's returns show with never-failing regularity, that with a fall of temperature there is invariably an increase of deaths from respiratory disease—phthisis, bronchitis and pneumonia. The true meaning of this is, I take it, not that cold is a great cause of respiratory disease, but that it is a great cause of death in those who are the subject of respiratory disease.

Now, we are accustomed to send wealthy patients suffering from incipient phthisis to the sunny south to pass the winter months, and without going into details as to the relative merits of this or that place, I will make a few general observations which may be of help to you in giving advice on this matter.

Now, remember that our object in sending the patient away is that he may breathe pure air, and we rightly argue that the amount of exercise which he will get in the pure air, will very largely

depend upon the amount of fine weather and sunshine.

But we must remember that in the winter, the hours for outdoor exercise extend from about 9 a.m. to 4 p.m., so that in the 24 hours there are about 7 hours in which the patient may be out, and 17 hours when practically he *must* be indoors. He will breathe the air of his house for twice as many hours as he breathes the open air. Therefore it follows that the selection of the patient's house or rooms is quite as important as the selection of the locality in which the house is situated. I may say at once that in my opinion there can be no worse place for a sufferer from chronic phthisis, than a monster hotel built tier upon tier, with overcrowded public rooms in which *poitrinaires* are often herded together in a manner which is most undesirable. I believe that the aspect of the patient's rooms is a matter of prime importance and one which is too often neglected. The bedroom should face the south-east, in order that the early visit of the sun may tempt him from his bed, enable him to dress in the warmth, and admit the fresh air by opening the window, more frequently than otherwise would be the case.

South-east is admittedly the ideal aspect for a house, but I would say that for patients visiting the Riviera, a south-east aspect is almost essential because of the virulence of the dry, cold, north-west wind, or mistral, which is very trying for invalids. If the windows of the rooms occupied by the patient look to the south-east, he gets the whole benefit of the morning sun, with absolute protection from the mistral, which, when it blows with violence, is far more keen and cutting than any of our east winds here.

A word of caution must be given as to allowing real invalids to participate in the gay social life which is rather too common in health resorts which have become fashionable. When last I was in the Riviera, I remember encountering a young American who was suffering from laryngeal phthisis, and who, instead of basking in the sunshine, spent the greater part of his days in the rooms at Monte Carlo, and of course was getting far more harm than good from his expensive journey to Europe. I think it is most important to lay stress on the fact that, for those who are suffering from incipient phthisis, all participations in crowded entertainments of every sort and kind should be absolutely disallowed.

(To be concluded.)

A CLINICAL LECTURE ON ACUTE (PARA-EPIPHYSEAL) OSTEO-MYELITIS.

Delivered at St. Mary's Hospital, Dec. 9, 1893, by

HERBERT W. PAGE,
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Surgeon to the Hospital; Examiner in Surgery, University
of Cambridge.

GENTLEMEN,—I have in my hand the notes of a case which was in the hospital in the summer, a boy who suddenly began to have acute inflammation of the lower part of his leg, and who lost in the course of a few days the whole of one fibula. He died very soon of pyæmia, secondary abscesses, as is common in pyæmia, being found in various parts of the body. An event like that is sufficiently tragic to arrest one's attention, even though cases of the kind are by no means uncommon, and may be very serious indeed in their results. We have in the hospital at this moment a girl who has lost nearly the whole of a clavicle; and not long ago I saw in the country the case of a child who has lost a considerable portion of the lower part of her tibia by exactly the same process of inflammation.

In the hospital I hear house-surgeons and others speak of the disease as acute necrosis; and the term is not at all an inappropriate one. When I was a student, such cases were commonly spoken of as "acute periostitis and necrosis;" but a better knowledge of the morbid processes in this particular inflammation has led us to do away with any suggestion that periostitis has really an essential part in the disease. It used to be thought that it was by the acute inflammation of the periosteum and the separation of the periosteum from the bone by the pus between them that the bone perished. But extended examination has now led to a different view of this particular form of inflammation, and we rather regard the inflammation of the bone itself as the cause of its destruction, and that the periosteum is, in the great majority of cases, only secondarily implicated. We look, therefore, upon the inflammation of the bone, the osteo-myelitis, as really the essential thing in these cases which one hears spoken of as acute necrosis; and in the particular instance of destruction of the fibula, which I have mentioned,

"acute necrosis" is by no means an unsuitable name to indicate what happened.

The extent and severity of the osteo-myelitis differ very much in different cases: the origin of it is not always the same; and there seem to be different kinds of cases at different periods of childhood. Early in life, within the first few months there is that form which has been fitly termed the acute arthritis of infants, described by Mr. Thomas Smith in the St. Bartholomew's Hospital Reports some years ago (vol. x). Then also you read of epiphysitis, of the acute osteitis of childhood, of acute necrosis, of panosteitis, and of the juxta-epiphyseal osteo-myelitis of the French; but whatever the varieties, and wherever the beginning, we shall be right, I think, in agreeing with Lannelongue, who says in his monograph on "Acute Osteo-myelitis during the Period of Growth" that all are merely osteo-myelitis.

To the various kinds one thing seems common: the tendency for the inflammation to begin in the immediate neighbourhood of the epiphyseal cartilage. Here, indeed, is one of the weak places of the organism; cell changes are continuously going on in the process of bone formation; vascular activity is working at high pressure in order to maintain the nutrition of a part where healthy nutrition is ever demanded, and the instability therewith associated apparently makes this part of a bone liable to be the seat of inflammation from causes often inadequate to produce such results elsewhere. "This zone of physiological proliferation," says Ollier, "is also the favourite site of pathological processes," and once inflamed the physical properties of the bone itself determine the violence of the inflammatory action: tension cannot be relieved by distension of the part; the vessels are soon blocked by inflammatory exudation, the spread of inflammation is encouraged, and death of the bone is the inevitable result. Should the inflammation spread in the direction of the diaphysis, the diaphysis may be entirely destroyed; or if it extends in the opposite direction the safety of the joint is gravely imperilled. Some of the forms of arthritis in childhood owe their origin to inflammation beginning and spreading in this way. It is to this particular class of case that the term epiphysitis has been applied; and although objections have been made to the name,—the chief being that there are doubts as to the inflammation really beginning in the epiphysis itself,—few will be found to deny that the growing

epiphyseal-cartilage line of a bone, whether long or flat, is a weak place, and that mischief of a most dangerous kind is prone to begin there.

At any rate it is the exception to meet with a case in which the central part of a diaphysis is alone involved. We had in the hospital not many weeks ago a case of that kind in which you saw me operate. A boy came in with the history of some mischief in the middle of the shaft of one femur, which had been going on for a period of something like two years. On making a free incision down to the bone we found there was an orifice in the thick new periosteal-made bone leading to the centre of the diaphysis; we enlarged the hole and removed a sequestrum; the wound healed up, and the boy has gone out perfectly well. Cases of that kind, however, in which the mischief involves the middle part of the diaphysis alone are extremely rare, it being the more ordinary rule for the mischief to begin close to the cartilage line at the epiphyseal end of the shaft.

I wish to ask your attention to-day to one of these varieties of osteo-myelitis beginning in the region I have named, with the special object of pointing out how the disease may be suspected and recognized at an early stage, even though it start at some depth from the surface, and the steps which may be taken to save, not the bone only by arresting the spread of the inflammation, but the joint also, even the whole limb, and the patient's life.

A very remarkable case of the kind was in the hospital some years ago, a case so appalling that it made a very great impression on every one who saw it, so great, indeed, upon myself also, as to lead, I hope, to very much better dealing with similar cases which have been in the hospital since. I do not intend to relate it in minute detail; but briefly, it was that of a little girl who, one rather cold summer afternoon, while playing on the pavement of one of the streets near, complained of a sudden pain in her left knee. This pain never afterwards ceased. She was brought here; and on admission intense tenderness was found on gentle pressure over the inner tuberosity and the adjacent part of the diaphysis of the left tibia. There was some swelling of the soft parts over this region, and a slight redness, with increased heat; but there was no fluctuation. The temperature was 104.6° F., and the child was obviously very ill. As she was worse on the following day, an incision was made over the seat of the most ex-

treme tenderness, but no pus was found. The next day there was rather less pain, and the temperature had fallen. The dressings were changed and a small quantity of curdy pus now came from the opening. Six days after admission, and eight after the onset of her illness, she complained of pain in a similar position in the other leg, in the right tibia; but there was neither redness nor swelling. The wound which had been already made on the left side began to be covered with a thick diphtheritic-looking membrane, on the removal of which pus flowed away, and on the passage of a director beneath the periosteum below the epiphyseal line, that is to say, over the diaphysis, more pus was liberated. Notwithstanding this, the tenderness, etc., increased, and a longitudinal incision had to be made right over the diaphysis through the periosteum. Further tenderness and swelling appeared; while the mischief now began afresh in other places, viz., over the internal malleoli of both tibiae. An incision was therefore made on the right leg through the periosteum; but no pus was evacuated. The further history of the case is one of recurring disaster. In spite of every effort to procure adequate drainage by openings in the popliteal spaces of both limbs, the periosteum became more and more separated from the diaphyses, and both knee-joints became distended with fluid. The right joint was found by aspiration to contain sero-pus; it was accordingly laid freely open, and continuous irrigation was begun. Nevertheless, the suppuration spread; and after the child had been in the hospital a few days more, when she first came under my own care, it was necessary to obtain the sanction of the parents to amputate the right leg as the only apparent means of saving her life. She immediately began to improve, the temperature fell to 100° F., and the inflammation in the other knee began to subside: but, presently, as indicated by increased swelling, redness, suppuration, and pain, the inflammation and death of the diaphysis steadily extended, and the child again began to fail. As it seemed quite useless under the conditions then present to remove the whole necrosed shaft, we urged that the child should lose her left leg also, as a means of saving her life. The parents, however, steadily refused to give their sanction; and the only course open to us was the removal of the diaphysis of the tibia on this side. It was picked out piece by piece, the periosteum being carefully left wherever it could be seen.

After this the temperature fell, and the patient very soon began to improve. Her life was saved, but only after the loss of one leg, and the loss of the tibia of the other, for the periosteum had been so much damaged by the acute inflammation of it and round about it that its bone-forming properties were practically destroyed. The parents have withheld their sanction from any further attempt at bone-grafting, and the girl is now a helpless cripple.

Examination of the tibia of the amputated limb revealed the following conditions, and I have here a drawing by Mr. Ridley in which they are shown. The bone is sawn through longitudinally, and the inflammation is seen to be focussed at the upper and lower ends of the diaphysis, at that part which the French have named *juxta-epiphyseal*, but for which the term *para-epiphyseal* is etymologically more correct. The whole upper epiphysis is loosened from the shaft, and beneath the cartilage are several small sequestra embedded in pus. This focus of suppuration has extended upwards into the epiphysis itself (the central portion of the epiphyseal cartilage having been absorbed), and through the right half of the epiphysis by a narrow sinuous channel into the knee-joint, having perforated the right articular cartilage, in which a small hole may be observed. Burrowing also outwards along the line of the epiphyseal cartilage, the pus has reached the periosteum, and denuded a considerable portion of the upper third of the shaft. The medulla of the middle third, or more, of the diaphysis seemed to be healthy; but at the *para-epiphyseal* zone at the lower end of the bone, the same process as at the upper end was obviously at work, and the epiphyseal cartilage has been partially absorbed by the inflammation on its way to invade the epiphysis. With the microscopical appearances I shall not trouble you. A full account of them by Mr. Silcock is to be found in vol. xxxix. of the Transactions of the Pathological Society, and the actual specimen is preserved in the museum. The drawings show you how there is this zone of central inflammation in the diaphysis immediately contiguous to the epiphyseal cartilage, far from the surface, and the minute channel by which the mischief had spread up and perforated the articular surface, and so gained access to the joint.

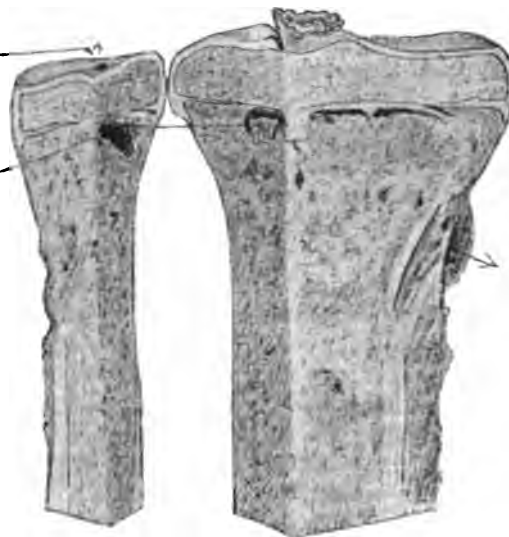
Precisely the same thing as is seen in these drawings was found to have happened in the case of acute necrosis of the fibula, which I referred to at first. There we found on exploration that at the malleolar end, close to the cartilage, there was



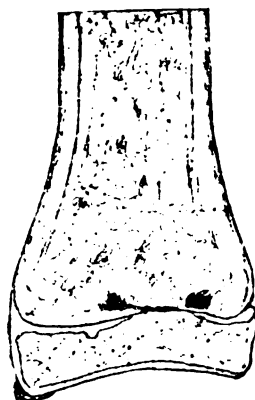
The covering cartilage showing minute opening into the joint.

Perforation of cartilage into joint.

The central para-epiphyseal necrosis and absorption of epiphyseal cartilage.



The sub-periosteal opening.



Lower end of diaphysis with central osteo-myelitis.

acute inflammation and destruction in the centre of the diaphysis of the bone, that the same thing had happened at the upper end, and that the inflammation, spreading upwards from the lower and downwards from the upper part, had invaded the

whole diaphysis, and thus led to its complete destruction. The periosteum was, moreover, entirely separated from the shaft by the amount of pus which had burrowed underneath it, having made its way there from the centre of the bone.

These two cases are sufficiently striking, and I should like to take them together as a text, from which to draw lessons for future conduct.

Note, in the first place, the insidious origin of the inflammation: there was no history of injury, and it may well be doubted whether the exposure of the child while playing on the pavement, could have been sufficient by itself to determine the onset of so terrible an inflammation. As has been abundantly shown by numerous experiments, the process is essentially a septic osteo-myelitis; but it is often difficult, if not impossible, to say when or how the living contagium finds an entrance to the system, and meets with a suitable place wherein to do its work, at some naturally weak spot such as we know the epiphyseal growing line to be. It is, of course, possible that exposure to cold may be sufficient to lessen still further the resisting power of a part which is inherently weak, so that an exceedingly slight septic contamination can start the inflammatory process, or what comes, no doubt, to very much the same thing, the patient may be already in a septic state when such a trifling injury as exposure to cold disturbs the equilibrium of healthy circulation and growth. The tendency shown by persons engaged in certain occupations to have their wounds—even subcutaneous wounds—immediately suppurate, may be explained, I think, by the fact that they continually work in a septic atmosphere, and their tissues are already septicly infected before the injury is received. We had a remarkable case of that a little while ago in a boy who had a traumatic separation of the lower epiphysis of his radius. There was nothing in the way in which the injury had been inflicted to lead us to think that it would be more serious than ordinary cases of like injury; but, being a stable boy, an acute septic osteo-myelitis very quickly supervened, and rapidly spread up the bone, so that in the end he had to lose his limb. How different the termination in a case I saw the other day, where precisely the same injury had befallen a healthy child, living midst perfectly healthy surroundings, and where no further ill results have followed beyond the necessary confinement while the epiphysis was being re-united to the shaft.

Let it suffice, then, to say that in this form of acute osteo-myelitis beginning at the para-epiphyseal zone, there may be no such cause as a local blow; and in arriving at a diagnosis we must be careful to remember that no such history is essential,

The difficulties of diagnosis may be very considerable. There is the local pain, continuous and increasing; but some days may go by before there are any signs of inflammation; and if the mischief is far from the surface, the local visible signs of inflammation may be so slight as to be no real measure of the havoc which is being wrought within. But if these manifestations of inflammation be never so slight, the constitutional condition of the patient is such as to indicate that there is something seriously wrong. A local pain of seemingly small significance, or a local redness which one might expect to subside after a few days' rest and a hot Boracic fomentation, is not associated with the serious phenomena which you see in these cases—the temperature running up to 104° or 105° , the rapid pulse, the wandering at night, the prostration, the thirst and dryness of the tongue. Obviously, there is some grave septic mischief going on; and it behoves us to take note of these concomitant constitutional symptoms as a means towards the accurate diagnosis of those cases in which the mischief has only recently begun, and there has not yet been time for any great extension of the osteo-myelitis. Add to these things the fact that the site of the pain and the exquisite tenderness coincide with the site of the weak place in the growing bone, and I think we shall often find that there are enough symptoms to lead to a correct diagnosis. Not always, however, and I can recall to mind a case in this hospital several years ago where the existence of osteo-myelitis was altogether unsuspected. A young lad had been for some weeks in the medical wards under the belief that he was suffering from typhoid fever, other explanations of his illness having been carefully excluded. Not till six weeks had gone by did local tenderness and pain about the upper part of his thigh point to the desirability of surgical advice. On seeing him, I explored beneath the periosteum, but found no pus. Deep-seated mischief, however, was suspected in the bone, and at the post-mortem examination, in the course of a few days, we found that, in the upper part of his femur, there had been the very condition I have been describing to you.

Diagnosis, therefore, is not always easy, and you see how many points have to be taken into consideration in arriving at it; but it is of vast importance that it should be arrived at soon. Recall the history and the results of the osteo-myelitis in this little girl, and in the boy whose fibula was

destroyed; recall, also, the results of other cases which have come to the hospital after the chief damage had been done, where the only thing left was to remove the limb or, by a tedious operation, to get rid of dead bone; and you can see how important it is to arrive at a diagnosis as soon as possible.

Let us look again at the drawing of the tibia in this particular case. We notice that although the mischief has begun at one particular spot, nevertheless the pus, which, of course, must try to find exit somewhere, makes its way in one or other of three directions. It may go towards the surface, and there collect and increase beneath the periosteum, which it promptly proceeds to strip from the shaft. More dangerous still, it may burrow across the epiphyseal cartilage into and through the epiphysis itself, through the covering cartilage right into the joint, and there light up an acute septic arthritis, for which in all probability there is one end, and one end only. The third route is down the diaphysis itself, spreading mischief as it goes, and leading to further destruction of the shaft, so that in the course of a few days the whole diaphysis may be destroyed and, bathed in pus, lie loose in the periosteal sac. Now, if the surgeon can imitate the least evil and dangerous of these three routes, in other words, if he will make an opening into the centre of the bone, he has it in his power to arrest the further progress of the inflammation, provided, that is, that he does it early and does it thoroughly. His diagnosis made, he must there and then trephine or gouge into the centre of the para-epiphyseal region of the diaphysis; and he must not be diverted from this course by the discovery of pus beneath the periosteum. It is of very great importance for you to remember this; because in the days when these cases were spoken of as acute periostitis and necrosis, it was thought sufficient to make an incision through the periosteum and let out the pus: nothing was done to the bone, and in consequence very many patients died of pyæmia, the bone itself never having been examined at all. Nor must he be deterred from going on by the absence of pus when he makes his incision through the periosteum, in the belief that he has had to treat a case of acute periostitis. It may be questioned whether there is such a thing as acute periostitis in this region apart from inflammation of the bone, or at least, it is extremely rare. We have seen in the case of this girl how entirely local may be

the original osteo-myelitis, and I say so even though, at first glance, it may appear to be otherwise, because of the affection of the second tibia and the lower epiphyseal portions of both bones. These inflammations occurred later, however, and may be regarded, I believe, as pyæmic and secondary to the inflammation of the tibia originally involved. At any rate, we shall not be unwise to take this view if it leads us to attack the primary mischief as soon as possible, and so prevent the same thing arising in other bones. Prompt action is above all things necessary; and this was the lesson forcibly brought home to us by this particular case. An incision must be made right down to the bone at the site of the surface inflammation, of the local tenderness, of the local pain; and the bone must be forthwith perforated at the spot. Pus, it may be only a drop, will well up; all the softened and inflamed bone must be then gouged away; the sources of infection must be removed by scraping and irrigation; the cavity formed must be thoroughly cleansed, and repair will take place by granulation in the usual way. The necessity for this has long been recognized. In 1865 the lower end of the femur was trephined by Ollier, who has done so much for bone surgery, but his example was rarely followed for many years, and his line of practice is not sufficiently enforced in our text-books. There has been a dread of opening the cancellous tissue of bone, and hesitation in doing so at the right moment whenever there seemed any doubt as to diagnosis, so that many bones and limbs and lives have been sacrificed which might otherwise have been saved. But in these days of perfect cleanliness in surgery, the surgeon need not fear to make a hole into the centre of the bone, and we can all agree with Ollier when he says:—"Antiseptic trephinations do not lead to necrosis; on the contrary, they are the best means of preventing it."

Let me now mention one or two cases in which we have endeavoured to carry out the line of treatment which I have described, and, as we believe, with the very best results.

A little boy came here one April, complaining of pain at the right internal malleolus. A few days before he had noticed a slight pricking sensation at the part, but it had not prevented him walking or playing about as usual. He had had no injury. Four days before he came into hospital he awoke with a much more definite pain at the inner side of the ankle-joint. Very soon there was swelling

of the part; redness followed, and this, together with the swelling and pain, decidedly increased during the next three days, so that when he came to the hospital there were obvious signs of acute local inflammation with some effusion in the ankle-joint. Moreover the least movement distressed him. The temperature was 103° F., and he was unquestionably very ill. On my arrival at the hospital in the afternoon I had him taken to the theatre at once; an incision was made above the internal malleolus, and a small quantity of pus was let out from beneath the periosteum, but because of the hesitation which I have been condemning I did no more. Mark the consequences. The relief afforded by this incision was practically nil. After the lapse of three days the boy was again placed under an anæsthetic; and there being no evidence of further periosteal mischief, I proceeded to do what should have been done before, and made an opening with a trephine into the centre of the bone at the lowermost end of the diaphysis. Pus welled up at once; the hole was enlarged, and I was thereby enabled to scrape out the inflamed and pus-saturated medulla from the lower two inches of the diaphysis. Further, for the purposes of drainage and washing out another opening was made higher up, and Perchloride solution sent through. We got rid of all the inflamed medullary tissue, there was no further extension of the osteomyelitis, and loss of the diaphysis, suppuration, and involvement of the ankle-joint were absolutely prevented. Some minute fragments of dead bone were from time to time spontaneously detached from the margin of the hole, but the periosteum resumed its natural position, and the cavity in the bone was gradually filled up.

In three other cases shortly afterwards, I had the opportunity of arresting the spread of osteomyelitis by freely gouging and scraping; and as an evidence of the success of the treatment, although in two of them the ankle-joint had already been invaded, and it was necessary in addition to lay the joints freely open, both healed soundly, and recovered useful limbs, and in neither was the joint completely ankylosed. Another case, showing that the mischief may occur in bones other than the long, was one in which the crest of the ilium was involved, resulting in an abscess which tracked down the thigh, and which in many of its symptoms very closely simulated acute inflammation and suppuration of the hip-joint. Only three weeks ago I had a case like those I have just men-

tioned—the case of a little girl whose symptoms and general condition were first of all described to me by the doctor under whose care she was. I said I felt sure there was some acute mischief in the bone, and that the proper thing would be to make an opening right into it with a gouge. He did so, but not with sufficient thoroughness, and the temperature still continued high and rose to 104° F. I was then taken to see her, and found the whole leg cedematous, the child extremely ill, and already some effusion in the ankle-joint. The opening was forthwith enlarged, and from the lower third of the tibia a quantity of acutely inflamed medullary tissue was gouged out of the centre of the shaft. A counter opening was made for the drainage. Although that was only three weeks ago the child is already on the high road to recovery. The temperature has been normal; one of the openings has already healed, and the other is closing up fast. There can be little doubt that if this operation had not been performed, the child would have lost its limb, and might not even have been alive at this moment. In a recent number of Langenbeck's Archives, there is an instructive paper by Thelen on this very subject. He points out how surgeons have been prone to rely on expectant treatment, and with what disastrous results, for he does not doubt that the tendency of other bones to follow, in the course of a few days, the example of the one in which the mischief has begun, is due to the infectivity of the primary osteomyelitis. He records the case of a boy, aged 10, who was suddenly seized with a pain in the right knee, and who came under observation on the fourth day after the onset of the symptoms, being then very ill with high fever. Near the patella there were heat, redness, and swelling of the skin, with acute tenderness on pressure over a spot at the lower end of the femur. He incised the periosteum, but found no pus; whereupon, with boldness and judgment which we shall do well to imitate, he trephined the bone, and let out pus from the centre of it. There was a rapid subsidence of all the symptoms, and further spread of the mischief was stayed. In a second and third case, as in that of the little girl which I have just related, the opening he made was too small, and the operation had to be repeated in the course of a few days.

The experience of several other surgeons points in exactly the same direction. I shall, therefore, not trouble you any more with a consideration of

the question, or of the different names which have been given by different surgeons to this particular form of inflammation of bone. The name is of no earthly moment. The important facts are that the epiphyseal ends of the diaphyses of the long bones are places where acute and dangerous osteomyelitis is prone to begin, and that there is one way, and one way only, of dealing with it successfully—by the early and free use of the trephine or gouge with which to go down into the centre of the diaphysis, so as to extirpate every particle of tissue which is diseased.

CLINICAL NOTES.

(Specially reported for The Clinical Journal. Revised in each case by the Author.)

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**WITH DR. W. ALLAN JAMIESON
 IN THE OUT-PATIENT CLINIQUE FOR
 DISEASES OF THE SKIN,
 EDINBURGH ROYAL INFIRMARY.**

Case of Tinea Barbæ of first grade.

The patient before you is a man of middle age, engaged in the Post Office. A month since, he submitted himself to the tender mercies of a barber, and was shaved. Three days afterwards he began to suffer from a slight itchiness on the left side of the chin, and this attracted his attention. When he looked at it he found there was a little reddish margin, crescentic in form, and there followed a slight eruption. He remembered he had a brother-in-law who had ringworm of the wrist and legs, and who had got a prescription for this, which he applied to the part without doing it much good. The condition extended, and new foci of the disease became apparent further down the neck, but you will notice, as he comes to show himself, that the condition is quite superficial. Now, when ringworm affects the beard it may simply be a superficial lesion affecting the epidermis, and corresponding to what you see in ringworm of the non-hairy parts of the body. The fungus does not penetrate into the hair follicles, and affecting the epidermis it expends its greatest activity at the margin of the part. As the fungus grows, as the little bit of mycelium, for really it is not a spore, becomes

implanted in the skin, it sends out hyphæ, and these insinuate themselves between the horny layers of the epidermis, splitting them up, and probably, by producing an acid, or, as others think, a toxin, sets up the sensation of itching. We know that some of the mould fungi do produce acids, and we know that the fungi that produce ringworm are mould fungi or are allied to them.

It itched, and then his attention was directed to it, and the disease spread at the edge, the parasite causing a little hyperæmia at the points where it insinuates itself under the newly-infected epidermic layers. Apparently there is a part behind this, within the advancing ring, which becomes protected from the influence of the fungus, or the reaction of the fungus is less, because the skin of this part is less red or almost normal, and that is very well marked here. You have here a red erythematous margin and a condition within which is nearly normal. The hairs here are not affected. Sometimes, however, the whole surface is scaly, red, and dry. I would ask you to observe the difference between ringworm of the beard in men, and ringworm of the head in children.

In the latter there is no erythematous edge to the eruption, though this is described by some authors. There is little more than scaliness, either yellowish or greyish, and brittle or broken off hairs; but on the beard we have an erythematous edge with or without a scaly centre, the hairs being little affected. That is the first and most simple form in which it affects the beard, but in general it affects the hairs. It sets up a folliculitis, the follicles swell, a sort of glutinous material is poured out, and then pustulation occurs, probably owing to the presence of pyogenic organisms as well. The hairs become loosened and fall out. That is the deeper condition of this, a condition in which there is a round patch with a distinctly swollen appearance, studded with inflamed and suppurating follicles, and with a number of hairs already fallen out or that can be extracted with ease. A further process occurs; the subcutaneous tissue swells, the fungus penetrates down into the roots of the hairs, and then you have a nodular elevation which seems like an abscess, and when you press with the finger you get semi-fluctuation. If you plunge a knife in you find no abscess. It is simply an œdematous condition of the skin, and

gives rise to great disfigurement. The nodular elevation may be all over the beard and whiskers, but very seldom on the upper lip. The upper lip seems to remain immune in the large majority of cases. These are the three forms. The first superficial; the second a deeper condition in which crusting, falling out of hairs, a general swelling of the skin, and a dirty unhealthy sort of appearance is produced in consequence of the oozing and crusting; the third, the nodular or bossy formation where the subcutaneous tissue is involved as well. According to M. Sabouraud, who has devoted a great deal of attention to this, the fungus that produces ringworm of the beard is a form of the trichophyton, and he calls it the trichophyton megalosporon because the spores are of a diameter of seven to eight micro-millimetres, that is, they are near the size of red blood corpuscles; whereas there is another form, which is met with on the head in children, in which there is a fungus producing spores which are only three micro-millimetres in diameter. In the large-spored form you have evident mycelium. In the small-spored form you have no mycelium, or it is very delicate and can't be made out so well. Where is this micro-organism derived from?

In some cases it is derived from animals, from horses, or from cattle, and we meet with ringworm of an intense character in farm servants, milkmaids, and grooms, due to the direct connection with horses or cows; but in town life this source of the disease is rare. We must look for some other, and although we have not yet traced the chain, we have distinct reason to believe that this fungus, derived from one of the moulds, is a mucor. How it assumes that particular character, which enables it to thrive on a living tissue, at least on a tissue which has a certain amount of vitality—the horny layers of the skin and hairs growing in the skin—while its previous existence has been on dead matter, such as any substance of a moist character which undergoes decomposition, as on bread, or jam, if you lay it aside for any length of time, we don't know. Mould grows on any substance which is damp, and placed away from the light for a time. It is called a saprophyte, from the Greek *σάπρον*, decayed, and *φυτόν*, a plant. This saprophytic stage of the trichophyton has so far eluded observation. It is quite possible this change may arise in the barber's shop in the following way. The barber sets aside his brush in his soap dish in a corner, after he has employed it in shaving. It

lies in a moist condition in the soap. There is a certain amount of epidermic scale on the soap on the brush, and this would very easily form a situation in which the mould fungus may acquire the property essential to its growing as an epiphyte. It is difficult to see how all these are cases of direct infection. Some are, because barbers sometimes shave a number of people and all have become affected one after another with ringworm. The fungus is lingering in the soap brush, and is implanted by the edge of the razor; but how does it get there? The idea was that the barber was shaving the head of some child who was suffering from ringworm, and in consequence of that his brush had got contaminated, and had produced this disease on the chin of the man who was shaved afterwards. Even if that were the case, since the large proportion of children affected with ringworm show the small-spored variety, while in ringworm of the beard the large-spored variety is found, this will scarcely explain the fact. The small-spored variety, as far as we know, can't be directly changed into the large-spored variety. Only a few children are affected with the large-spored variety, and it could only be from these that infection could come; so I think we must not blame the barber for want of care in cleaning his brushes. Probably it is not communicated from the heads of children. It is more likely this mould fungus undergoes a peculiar change that renders its power of growth on skin a possible one. We often meet with ringworm of the body in children and adults on the covered parts which are not exposed, except when undressing, to the action of the air. A child is brought with ringworm of the arm or shoulder, and there is no history of contagion. We find it is ringworm. How did the child get it? We can only account for it on the mould theory—that in some way the flannel or underlinen worn by the child had been lying away in a slightly damp condition; that this became impregnated with epidermic scales, and in that semi-moist condition the mould fungus underwent the change which enables it to alter its habit of life from saprophytic to epiphytic. I think that is the explanation; we will find it out in time.

This man has come to us to get cured. How are you to cure him? You are to cure him by keeping the part clean, washing away all epidermic scales, and, secondly, employing some remedy that will act on the fungus. Fortunately, it is on the surface and should not be difficult to cure. If you

use an ordinary alkaline soap in washing this man's chin, you produce an effect which is commonly seen in children or females who have delicate skins; you produce a scurfy condition. Alkaline soap withdraws the natural oil so that the outer epidermic layer dries up, cracks, and becomes slightly scaly, and thus forms the best situation for the growth of the fungus. Therefore, don't use ordinary soap for the purpose. Employ a neutral soap or, preferably, one containing a slight excess of fat, and a soap of which the alkali is potash is preferable to one in which the alkali is soda. A certain amount of decomposition occurs in all soaps when mixed with water, and a certain amount of alkali is set free; but this is minimized by using a super-fatted soap. That is the first thing, to wash it with this soap. What is to be the parasiticide? At present Mercury enjoys some reputation, although I think it is rather on the wane as a parasiticide. I have not been able to satisfy myself that Perchloride of Mercury is at all valuable, but there are Salts of Mercury which are useful. The Ammoniated Mercury is of value, but probably it acts as a parasiticide not so much on the trichophyton but on associated pyogenic organisms, because we know that Ammoniated Mercury in small quantity lessens the activity of these pyogenic organisms. We see this in impetigo contagiosa especially, and in sycosis. The most valuable remedy is Sulphur. Sulphur, when applied to the skin, has a distinctly antagonistic action on the growth of these lower fungi. That has been long known in other regions besides the skin. It has been employed by gardeners for the diseases of plants due to parasitic organisms, and has a well-deserved reputation. We combine a little Sulphur with Ammoniated Mercury, and we must mix it with some excipient, and the best is Lanolin. Lanolin, although sticky, is not prone to decompose, and is now refined so much that it forms a suitable basis. I don't think we can make much of the fact, that as being prepared from sheep's wool it is the natural oil of the skin, that it is a cholesterine fat instead of a glycerine fat. The little tendency it has to become rancid, the stickiness to adhere to the skin, and the property of absorbing water so that it can be rubbed up with water, constitute its chief advantages.

R. Sulphuris Sublimati ... 3j
Hydrargyri Ammoniaci ... 3ss
Lanolini 3j

This is rubbed into the part twice a day. With

that method of treatment you can state positively that in ringworm of the body you can cure it in a short time; and in ringworm of the beard you can cure it, but not in quite so short a time, because the difficulty is for the parasiticide to act closely and intimately on the part affected, and the deeper the seat of the disease the greater difficulty you have. That is the difficulty you have in curing ringworm of the head in children.

Prurigo with Chronic Eczema, closely resembling Prurigo Hebræ.

This is a boy whom I have brought down from the wards. He was admitted two days ago, and has been subjected to a certain amount of treatment. Now, the first thing you notice is how thin the boy is; the legs are wasted, the skin is very dry and it is sallow, not the clear hue of the healthy skin of a healthy boy. He has a number of red patches, which are due partly to scratching, partly to congestion of the skin, and partly to the morbid degree of induration which has taken place. Were this boy to live in Vienna instead of living in Scotland, he would be shown as an example of prurigo. This would be a typical case of prurigo in Austria. It commenced in infancy. That is the reason why it is so dry, and why those thick eczematous patches are on the surface.

The limbs are particularly affected. That is the case in prurigo, but a difficulty would arise. It occurs in the ham as well as on the outer aspects. The ham is just the place, and the bend of the elbow, where they say in Austria that prurigo does not occur. This is one difficulty in the way of regarding this case as prurigo in the Austrian sense. This boy suffers from intense itching, from a great deal of chronic eczematous condition and general dryness of the skin.

It is usually stated in Austria that the condition is worse in winter and better in summer, and this is natural, because people there perspire freely in summer and the skin becomes softer in consequence. In their cold winter the skin perspires little. With us there is usually not so much difference between winter and summer, and the skin remains much in the same condition in both seasons; hence, perhaps, the explanation of the boy's statement that he is better in winter and worse in summer. He is, however, a young boy, and we cannot rely too much upon his answers. What are we to do for him? We must try to soften the skin

and to prevent the tendency to scratch. Now warm baths containing starch or starch and Boracic Acid, soothe the skin very much. These will be used at night, and after the bath an ointment containing β Naphthol will be applied as follows:

R Naphtholis β .
Zinci Oxidi āā 3ss
Lanolini.
Vaselini āā 3ss

This is to be rubbed in also in the morning. That cures the eczema and tends to diminish the tendency to scratch. Internally see that all the functions are acting well, and give him a good deal of fat, butter, cream, milk, and especially Cod Liver Oil. I think, in the course of two or three weeks, we shall have a considerable improvement in the condition.

Eczema e Pediculis.

In both of these cases, mother and daughter, we have the presence of pediculi on the head, and the pediculi are really the cause of the whole condition. It is the scratching which has set up the irritation in the skin, and has produced a secondary eczema as a result simply of the use of the finger-nail. What we have to do is not to cure the eczema, but to destroy the pediculi. The best thing for that, probably, is kerosine or paraffin oil.

R Petrolii Venalis 5vj

The head is to be soaked with this to-night and to-morrow, and to be covered with a cap. Keep her away from any light. Then wash the head well with soap and warm water. Remove the crusts with a comb, and treat the impetigo contagiosa with a weak Ammoniated Mercury ointment.

THERAPEUTICAL NOTES.

Carcinoma Cervicis Uteri.—Dr. J. X. Bernhart has used injections of Salicylic Acid in cases which are beyond surgical interference. He uses a 6 per cent. solution of the acid in a 60 per cent. alcoholic menstruum; of this about 3ss is employed for a sitting, a few drops being injected in six or seven places in the mass of the growth. A sitting takes place every four or five days. Dr. Bernhart claims that after a few sittings the discharge becomes much less, the pain diminishes very considerably, and the general condition improves in proportion. A condition closely resembling an actual cure was brought about in several cases.—(*Centralbl. für Gynäk.*)

Acne Rosacea.—Dr. Petrine recommends the following application:—

R Resorcin gram.j
Ichthyol gram.ij
Collod. Flexile gram.xxx

The pustules are to be emptied first, and then the above mixture painted on daily for three days; an interval is then allowed till the Collodion has peeled off, and then the process repeated as often as is necessary.—(*Münch. Med. Wochensch.*)

REVIEW.

A Contribution to the Pathology of the Vermiform Appendix: a Thesis for the M.D. Degree of the Victoria University. By T. N. KELYNACK. (H. K. Lewis.)

Graduation theses in England are but too frequently mere perfunctory compilations of excerpts from the work of others, and it is therefore with especial pleasure we note this most brilliant exception. The monograph before us reflects the greatest credit on its author, and does honour to the Victoria University: for extent of research and minuteness of detail it deserves all praise that is bestowed on it. The first seven chapters are devoted to an account of the normal anatomy of the appendix, and its position with regard to the peritoneum and adjacent structures: from the number of the author's observations and his references to the observations of others, these chapters must be accepted as finally decisive of the normal position of the organ, if normal be allowed in such a variable quantity. Pathological changes in the contents, walls, and environments occupy the next sixteen chapters, and a classification of them is given in Chapter xi., which, in the words of the author, we consider "pathologically correct, and clinically useful," and we hope to see it universally adopted in future. Chapters xxiv. and xxv., dealing with symptoms and diagnosis, form an excellent resumé of our knowledge on the subjects; the concluding chapter on treatment is most judicious and reliable, the author's remarks on Opium and other forms of *medical* treatment are very sound, while the indications for *surgical* aid are clear and concise. The bibliography with which the thesis concludes, seems to us practically exhaustive and invaluable for reference.

Clinical Lectures at the National Epileptic Hospital, Queen Square, on Wednesday afternoons at 3 o'clock, will be resumed by Dr. GOWERS on Jan. 31, and will be given alternately by Dr. GOWERS and Dr. BRIDGES.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 31, 1894.

A CLINICAL LECTURE ON THE TREATMENT AND MANAGEMENT OF PULMONARY TUBERCULOSIS.

Delivered at University College Hospital, by

G. V. POORE, M.D., F.R.C.P.

Physician to the Hospital.

(Concluded from p. 198.)

I am inclined to think that in this country there are few spots better suited for the residence of patients in the early stages of phthisis than our own dry bracing sunny south downs, always provided that there be sufficient shelter from the north. A good well-built warm cottage facing south-east and south, with a wide verandah, and situated on the slope of a chalk down, is an almost ideal dwelling for the phthisical.

There is no greater practical mistake than to send patients to distant health resorts if they are unable to bear the expense of obeying one's orders comfortably. To send persons away who are obliged to calculate the expense very narrowly, to send them from "home comforts" to spend the winter in some cheap "pension," of which overcrowding and underfeeding are the main characteristics is to court failure.

I am not going to discuss the merits of different health resorts, or dwell upon the distinguishing characteristics of the sea-shore and the mountain tops. The main object which the doctor has in view, is to enable the patient to breathe pure air. Some health resorts are being killed by their own popularity, which has increased the price of building land, and has led to the building of houses in too close proximity to each other. In a health resort every house should be isolated, and stand in a garden of its own. It is well to bear in mind the difference between a holiday resort and a health resort. As a rule, we shall find, that in proportion as a place grows in popularity with "trippers" and holiday excursionists it becomes undesirable as a residence for real chronic invalids.

So far my advice has been to encourage persons

who have a constitutional tendency to tuberculosis, or who may afford actual evidence of the disease in its early stages, to select callings and professions and to live in places which will give them a fair chance of throwing off the disease, and give them as few opportunities as possible of their favourable "soil" being frequently re-seeded by the bacilli which are likely to be present in the air of houses or places to a degree bearing some proportion to the density of population.

But, it will be asked, can we do nothing to help the individual except by attending to his surroundings? Has the discovery of the bacillus tuberculosis given us no clue to some method of destroying the microbe *in situ*?

The answer to this question is very doubtful, and mainly for the reason that we are unable to get away from that stumbling-block of scientific therapeutics known as the *post hoc* fallacy. If, when a patient with phthisis is removed from the foetid atmosphere of some wretched hovel where he has been half starved and overworked, to the comparative purity of our hospital ward, where light, air, good food, and absolute repose are afforded him, we administer to him some remedy which has acquired a reputation for the "cure" of consumption, and if, in a few days time, his cough be easier, or his temperature lower, his appetite improved, or his weight increased, to which of many circumstances are we to attribute his improvement? Assuredly we have very slender grounds for attributing it to the drug. To a certainty the *patient* would attribute his improvement to the drug, for with patients, as a rule, *post hoc* is *propter hoc*, and belief in drugs is absolute.

When a drug rapidly produces some easily appreciated change in the bodily condition, we manage to escape from the *post hoc* fallacy. That purgatives cause diarrhoea, that antipyretics lower febrile temperatures, that Morphia causes sleep and contraction of the pupils, that Pilocarpine and Atropine influence the secretion of sweat are facts which nobody can doubt. When, however, we are told that the cessation of vomiting is due to the administration of Ipecacuanha in doses so small as to elude every known test we may accept the statement as a matter of faith, but we must admit that it is absolutely incapable of experimental proof

In the same way the cause of the improvement of chronic invalids whose condition is known to be for ever fluctuating is almost incapable of demonstration, although one is glad to be able to say that the improvement of myxœdema by the administration of Thyroid Extracts is apparently an exception to the rule.

It follows, therefore, that we must receive all statements as to the effect of drugs on such a disease as pulmonary tuberculosis with the utmost caution and reserve.

In discussing the treatment of phthisis by drugs I shall limit myself entirely to a consideration of those measures which have had for their object the destruction of the bacilli at the seat of their formation.

The use of inhalations of various kinds had been recommended and largely employed before the discovery of the bacillus, and since its discovery this line of treatment has had a fair trial. In many cases inhalations may have alleviated the patient's condition by somewhat lessening cough and secretions, but it cannot be claimed for any of them that they have had any effect in arresting the progress of the disease. When we consider how very dilute must be any antiseptic vapour which is capable of being inhaled into the lungs, when we consider that there is always residual air in the lung, and when we consider still further that only a small minority of the bacilli lie on the free surface of any cavity in the lung so as to be within reach of any antiseptic vapour which may be inhaled, we cannot be surprised at the failure of this mode of treatment to arrest the disease.

Koch's efforts to kill the bacilli by the injection of "Tuberculin,"—the product afforded by the pure cultivation of the bacilli—will be long remembered as one of the most interesting episodes in the history of therapeutics. Although the treatment was not destined to fulfil the sanguine expectations of the originator, the work which led to its promulgation was of the highest scientific value. The rapidity with which the profession throughout the world came to the conclusion that the dangers of employing Tuberculin were considerable, while the advantages were doubtful, was also creditable to the scientific methods of the nineteenth century.

The administration of antiseptics by the mouth has not been productive of any decided benefit. The Salts of Mercury and the Tar products have been used without decided effect. Among the

latter one must mention Creasote, which is just now going "out of fashion," after having been very much in vogue.

It seems to me that if any useful result is to be obtained by the employment of antiseptics in pulmonary tuberculosis, it is important to choose one which is eliminated by the lung—something which is absorbed from the stomach, mingles with the blood streams, and is eliminated by the lungs.

The bodies containing the active principle "allyl," such as garlic and onions, seems to fulfil these indications, and seem worthy of a trial. The most potent of these bodies appears to be garlic, for it is well known that the smell which garlic imparts to the breath is most persistent, and is produced by very small quantities. The allyl compounds such as Allylic Alcohol and Oil of Mustard were shown by Koch to be strongly antiseptic, and to have a remarkable power of inhibiting the growth of the bacillus anthracis. In this respect they rivalled the Salts of Mercury. If it be possible to hinder the growth of the bacillus tuberculosis in the lung, it seems at least reasonable to try the effect of such a body as garlic. But, it will be said, if garlic is capable of hindering the growth of bacilli tuberculosis, we ought to find that phthisis is less prevalent among garlic-eating populations than elsewhere. Facts are wanting to decide this point either one way or the other, but even should it be shown now or hereafter that there is no correspondence between the habit of eating garlic and a small death-rate from phthisis, it would not, to my mind, be a very strong argument against giving garlic a trial, for one could only suppose that such a method of treatment would be useful as a help when other circumstances were favourable. It could not reasonably be expected to be of any use in the face of overcrowding, starvation, dirt, and squalor such as are common enough in many of the cities of southern Europe.

Now, I think I may safely say that I have found garlic of distinct service in cases of dilated bronchi with foetid expectoration. In two such cases in which the expectoration was purulent and horribly foetid, we administered garlic in the food, until the odour of garlic was permanently present in the breath. In both cases the disgusting foetor of the expectoration was replaced by an odour, not foetid, but pungent, and reminding one in some degree of the odour of syringa. The change in the odour of the sputa was the same in both cases. The foetor disappeared, and was replaced by an

odour which was not that of garlic, but distinctly *sui generis*.

Mr. Ionides, who was my house-physician when one of these cases was in hospital, treated the sputa with Ether, and manufactured in this way a very pungent "scent," in which the syringa quality was distinctly present. In these cases the expectoration lessened, and the condition of the patient was distinctly improved.

Dr. Sidney Martin tells me that he also has given garlic to patients suffering from dilated bronchi and foetid expectoration, and with distinctly good results.

Then we have had two cases in the wards of boys who were apparently suffering from basic phthisis, and who made no improvement until they were placed upon garlic, when a rapid and complete recovery took place.

In private practice I can recall the case of a gentleman suffering from fibroid phthisis, who improved most remarkably, and gained weight very rapidly while he was taking garlic, which he did most persistently.

Another case was that of a girl with aphonia, who had very characteristic pyriform swelling of the aryteno-epiglottic folds, so that neither I nor Dr. Adams of Buckhurst Hill, who brought the patient to me, had the slightest hesitation in making a diagnosis of tubercular laryngitis, and giving the most guarded prognosis. The patient among other measures was ordered to take garlic. When next she came to see me a month or so later, the laryngeal condition had completely subsided. The result was so remarkable that one is inclined to doubt the accuracy of one's diagnosis, but nevertheless the result justifies one in trying the remedy again in similar cases.

I have usually given the garlic in the food. A "clove of garlic" has been chopped up and boiled with the beef-tea, and only very few patients have been unable to take it in this way. The evidence that the patient is under the influence of garlic is the persistent smell of it in the breath, a smell which is equally present when the garlic has been administered in the form of an extract, enclosed in gelatine capsules.

Garlic, I need say, is no new remedy. It formerly held a place in the London Pharmacopeia, but the extant British Pharmacopeia does not recognize it. I see that Dr. Brunton states in his work on pharmacology, that garlic is still in the Pharmacopeia of the United States. It is men-

tioned by Pereira and by most of the previous writers on Materia Medica. It was used by Hippocrates. Among the properties ascribed to garlic by old writers is that of a stimulating expectorant, so that its employment in lung diseases is no new idea. Its acrid taste and the disagreeable smell which it gives to the breath, have naturally militated against its becoming a very popular remedy. I have found, however, no difficulty in persuading patients to take it. Administered with food, it has never caused any serious gastric disturbance, the only trouble in its administration being a natural repugnance to the flavour and odour on the part of some patients.

The essential Oil of Garlic is said to contain a Sulphide of Allyl, and in this connection I would remind you that other sulphides, such as Sulphuretted Hydrogen, sulphur waters, the Sulphides of Calcium and Potassium have enjoyed a certain reputation in tuberculosis and "scrofulous" diseases.

Among other remedies which I think I have found serviceable in phthisis are the so-called "alteratives," such as Arsenic or Potassium Iodide in small doses, and the sulphides above alluded to.

Counter-irritation by means of Iodine or stimulating liniments, well rubbed in, until the skin of the chest is thoroughly reddened often have the effect of lessening the cough and expectoration.

Garlic is not only given off by the breath, but by the skin also, and I have been for some years in the habit of administering it to those cases, especially common in children, in which a cadaveric odour of the skin develops after attacks of acute specific disease. In such cases warm baths and soap are of no use, but I have found that, if garlic be administered until the skin reeks of it the cadaveric odour is got rid of, and is no longer noticeable, when the smell of the garlic disappears.

I have thus brought to your notice a body which on theoretical grounds seems worthy of a trial, and which from practical experience I feel inclined to continue to administer to sufferers from phthisis in its early stages.

As an Anæsthetic for the Eye, acting also as a Mydriatic:—

R. Cocain. Mur.
 Pilocarpin Mur. āā gr.iiij
 Aq. Destil. ʒiij

M. Signa: To be dropped into the eye.

The Clinical Distinctions between Stones Imbedded in the Renal Substance and those Loose within the Pelvis.

A Clinical Lecture delivered at the London Hospital, November, 1893, by

E. HURRY FENWICK, F.R.C.S.,

Surgeon to the London Hospital, Surgeon to St. Peter's Hospital for Urinary Diseases.

GENTLEMEN,—Whilst discussing the symptoms of renal calculus with you last session, I brought cases forward to impress upon you that renal colic does not only occur as the result of the passage of a renal stone along the ureter, but that other substances, such as blood clots, mucus-clumps, tubercular sloughs, hydatid cysts, and detached pieces of renal growth, were able to evoke in their transit along the ureter exactly the same colicky pain and distress, though, perhaps, in a milder degree.* I also mentioned that any sudden over-distension of the pelvis, either by water, pus or blood, induced typical renal colic. To-day I wish to go a step further, and whilst considering the subject of renal calculus in greater detail, to point out to you that certain forms of renal stones, *vis.*, those imbedded in the cortex, do not, as a rule, produce renal colic. Hence, renal calculi may be divided into two great groups according as to whether they are imbedded in the true renal substance or in a calyx, or whether they lie loose in the pelvis. Most of your text-books make no such distinction; but the importance of grouping renal calculi thus, is evident when we remember that not only do the symptoms differ in these two classes, but the prognosis also of the renal health in the two classes is widely diverse. My attention was first drawn to this subject by the very excellent lectures of Dr. Newman, in 1888, upon Surgical Diseases of the Kidney, and in 1889 by a pithy and pregnant addition to a discussion upon renal calculus by Mr. Bennet May.† My experience justifies me in accepting the statements which were then made. It is obvious on reflection that the symptoms caused by renal stones and the destruction they are able to produce, must, of necessity, depend upon their situation.

* Author, "Cardinal Symptoms of Urinary Disease," p. 215.

† Bennet May, "Brit. Med. Journ." 1889, pt. ii., p. 1084.

If they are enclosed in a cavity like the renal pelvis, sheathed, as that receptacle is, with sensitive mucous membrane, they must sooner or later irritate and inflame that surface, whilst their weight will cause them, from time to time, to fall upon the mouth of the ureter and produce those backward pressure changes which prove so fatal to the working capacity of the gland. On the other hand, if they are fixed in a deep calyx or are imbedded in the tolerant renal substance, their capacity for producing symptoms or for causing permanent and extensive damage is much curtailed. I have selected seven cases of renal calculus from a series of thirty operations on the kidney, which will serve to illustrate the differences between these two classes.

Fixed or Imbedded Renal Stones. The Cortico-Medullary Group.

Case 1. This man, who is 36 years of age, was sent to me by Mr. John Harris, of Dartmouth. He had been in several hospitals complaining of extreme renal pain, from which he had suffered for twenty years, but as his urine was normal, his sufferings were considered to be feigned or exaggerated, and nothing was attempted. But his pain was very typical in its character. It was situated in the kidney, and he could cover its position with the last phalanx of his thumb. It could be elicited also by percussion over the renal region, or by any succussion of the body. It came on with any exertion, even with walking. Mr. Harris also noticed that it was more severe if the quantity of urine passed in the day was small (12 oz.), but relieved if the supply was normal (48 oz.). The patient had never had colic nor any radiating pain beyond a left testicular pain if the renal suffering was acute. Sometimes he suffered from great frequency of micturition in the day, and occasionally had to rise five or six times at night, passing very little at a time. He had never noticed blood in his urine. The latter was clear, Sp. Gr. 1020, it contained no pus or blood, and only a few oxalates were visible under the microscope. The urine contained a slight amount of albumen. On receiving this account I sent a diagnosis of imbedded cortical calculus, and three weeks ago you saw me remove this rough pointed stone, which measures $1\frac{1}{4}$ inch in length and weighs $1\frac{1}{2}$ drachms. It was imbedded in the cortex of the lower end of the kidney. The wound healed, as these cases should, by first intention, and he is about to return home free from pain.

Case 2. I removed these three spiked oxalate of lime calculi from the cortex of the kidney of a patient referred to me by Dr. Macmillan, of Florida. The patient, who was 29 years of age, remembered suffering pain in his right kidney even as a boy, and since childhood the same pain has continued, though sometimes he has been free for several hours. If he ran or made any sudden movement, or committed any error in diet, the pain at once increased. When it was very severe it passed into the right testicle, but otherwise it did not radiate. No matter how severe the pain was at the time, it was relieved by lying on the affected side. This was so marked a feature, that he has always slept on the affected side, and if by chance he turned over in his sleep on to the opposite side, the pain awoke him and caused him to turn back again. He has never had colic. He has never suffered from frequency of micturition. His urine was 1020 in Sp. Gr. and quite clear, but contained blood casts and oxalate of lime crystals, and $\frac{1}{16}$ albumen. His urine varied in amount from 15 oz. per diem to 48 oz. The calculi were found in a hollowed out pocket in the cortex, which was situated on the posterior surface, very nearly on a level with the middle of the pelvis. The wound healed by first intention, and the amount of urine increased to 50 oz. per diem, and became stationary.

I have met with others which I could quote, but these will serve as type cases. I do not think sufficient care has been taken to ascertain and to mention the exact position of the stone found in these operation cases which are recorded in the literature, so that I cannot give you any idea of the proportion which imbedded stones bear to pelvic stones, probably they are in the minority.

Now let us glance at the better known class of loose pelvic stones. (Symptoms of typical renal stone described.) You will remember that a smooth uratic or smooth oxalate of lime renal calculus is often able to remain in the pelvis of the kidney for a considerable time before inducing those inflammatory changes which give rise to pus and phosphates in the urine. Sooner or later, however, these changes ensue and pyelitis is superadded. The stone then rapidly enlarges by phosphatic accretion, and may fill the pelvic cavity to the complete destruction by pressure of the tissue of the gland.

Case 3. I removed this small flattened oval calculus from the dilated pelvis of the kidney of a

patient of the late Sir Andrew Clark. He had suffered from violent recurrent colic for five years. At first the attacks were only once in six months, but the interval of rest diminished, until finally, when he consented to an operation, they took place every week.

Each attack was accompanied by numbness in the back, down the inside of both thighs, and in the calves of the legs and testicles. He had passed blood several times, but always of a mahogany colour. The patient had faithfully followed careful instructions of diet, and had taken courses of Piperazine without any effect. Before the operation the urine was 1010 in Sp. Gr.; it was acid, and contained $\frac{1}{8}$ albumen. The quantity was ample, he had never had frequency of micturition. I found the pelvis much dilated; but as there was a fair amount of cortex left, I washed out the kidney and drained it. It healed sluggishly, and left an obstinate sinus, which remained for months.

Case 4. I removed this conical calculus from the ureter of a patient, aged 30, who was sent to me by Dr. Warburton, of Pontypridd. His first attack of renal colic occurred seventeen years previously. He had passed phosphatic renal calculi for four years. The symptoms of ureteral block with suppuration commenced very suddenly, with repeated rigors. I found the pelvis full of pus, and this phosphatic calculus blocking the ureteral orifice. His wound healed sluggishly.

The amount of damage which can be done to the kidney by a small stone if it rolls loosely about the pelvis and falls from time to time upon the mouth of the ureter is remarkable. I could not show you a better example than this young fellow who was sent over to me from the medical side by Dr. Kidd.

Case 5. I operated upon him in April last. For many years he had been suffering from recurrent attacks of severe renal colic, and latterly a renal tumour had appeared in the left loin. On cutting down upon his kidney it was found to be transformed into a thin walled sac containing over a pint of odourless clear urine. I could feel no stone, and finding the layer of renal tissue which remained to be extremely thin, I removed the kidney. In it I found this little flattened oxalate of lime calculus. It is exactly like a small French bean and it only weighs two grains, yet by acting as a ball valve and frequently dropping into the mouth of the ureter, it was able to totally destroy the kidney by the backward pressure it exerted.

To accentuate the difference between imbedded and loose pelvic stones, let me draw your attention to this renal calculus.

Case 6. It is composed of oxalate of lime; on one side of it it is spiculated with fine glass-like crystals, but the other is covered with a layer of fibrous tissue. I removed this a month ago from the pelvis of the kidney of a man *æt.* 38. Sixteen years ago he commenced to suffer from renal colic; the attacks becoming more frequent and more severe as time went on. Eight years ago he took some "electric" pills (at that time largely advertised as a universal panacea). Gradually the pain ceased, and for eight years he has been entirely free from all symptoms. Two months before he came under my notice, he had a sharp attack of epidemic influenza, and on getting up the old renal pain recommenced, and he was seized with an agonizing colic. Day by day the renal colic recurred (quite unchecked, I may add, by a fresh supply of his old friends the "electric" pills), until he was at last in despair. I operated at once, for I had no doubt that a loose pelvic calculus was present. Probably it had hollowed out a cavity for itself, and had become imbedded in some calyx, about the period when he first took the pills, and thus it had ceased from troubling, until it had been probably loosened by the high fever of the influenza. When I opened the pelvis I found this stone loose in the cavity. The facts permit us to surmise that this fibrous looking layer on one side of this calculus marked the method in which it was glued into the calyx. The patient made an excellent recovery.

The striking feature in which these cases differ lies, as far as my narrow experience extends, is the difference in the character of the pain. In the imbedded stone there is constant and often acute pain, located in the kidney, rarely radiating unless it is very severe, and then to the testis of the corresponding side. Colics are usually absent. In the loose pelvic stones we encounter the typical renal colics with the radiation to the shoulder and leg.

There is usually also, I believe, a higher specific gravity in the urine of an imbedded stone than in that secreted by one which evokes intermittent colics. Probably the backward renal pressure exerted by the ureteral obstruction would account for this difference. *Albumen* and *blood-casts* are found in these cases, and the patient is sometimes diagnosed to be suffering from Bright's disease;

or the surgeon objects to operate because of the presence of albumen and casts. The latter, I believe, proceeds from the kidney in the immediate neighbourhood of the stone, and should not deter us from operating.

Cortical stones differ somewhat in appearance from pelvic stones. The former are usually composed of oxalate of lime, are of a dark brown colour, often encrusted with the finest glass-like crystals, and not infrequently they are of a pyramidal shape, or like a heart or double-fanged tooth. If the calculi are multiple, small flat greyish stones usually accompany larger rounded and spikey stones. Pelvic stones, I suspect, are more often uratic or urato-phosphatic, and are probably smoother and of an irregular shape.

The prognosis of the kidney varies very greatly in the two classes.

The kidney will tolerate an imbedded cortical calculus for years; the secreting structure, except in the neighbourhood of the calculus, does not suffer. On the contrary, the presence of pelvic stones are fraught with danger to the health of the kidney. Probably each attack of renal colic adds its increment of change to the backward pressure absorption which is seen in these cases, whilst the tendency they exhibit to inflammatory conditions of the mucous membrane is a standing menace to the contiguous secreting structure.

Prognosis of operation. This coincides with the statistics published by Dr. Newman, who was the first to draw attention to the effect of suppuration upon the death rate. He has collected 42 cases of nephro-lithotomy undertaken when there was no suppurative change. This series was without any mortality. When suppuration was co-existent, however, the death rate was 43.3 p.c. Cortical stones afford capital results. With due asepsis, with gentleness in removal and the employment of deep sutures the drainage tube can be withdrawn by the second or the third day, and the entire wound should heal by first intention. The dressings are usually wet for the first few hours, then dry; showing that the pelvis proper has not been opened. Blood is usually seen in the urine for a day or two, but this can be accounted for by the bruising of the outer wall of the pelvis and by the occasional rupture into the pelvis whilst extracting the stone. The operative result, on the other hand, of loose pelvic stones depends a great deal upon the condition of the kidney. Usually, if the stone has

been taken early, before colics and backward pressure have enlarged and inflamed the pelvis, the wound heals as readily as in the imbedded variety, but if dilatation and pyelitis is present a sluggish sinus remains.

The question of operative interference in Renal Calculi. It has been epigrammatically said that the presence of a large bladder stone is somebody's fault. There is but little doubt but that the same dictum will eventually hold for renal calculi. By all means let therapeutic and dietetic measures be conscientiously tried, but when these have had a fair trial the aid of surgery should be summoned, and this before the advent of pyelitis. Skilled exploration is almost free from risk. Operative interference should, perhaps, be sooner resorted to in those cases in which recurrent colic is suffered from than in those where pain of a localized character is complained of, that is in those in which fixation or imbedding is present or has taken place.

A LECTURE

ON

CANCER OF THE CERVIX UTERI.

Delivered at St. George's Hospital, Jan. 10, 1894.

By W. R. DAKIN, M.D.,

Obstetric Physician to the Hospital.

GENTLEMEN,—I propose to-day to consider the subject of cancer of the cervix uteri, a very common disease, and one with which you will often have to deal. The cases will come before you either before they are too far gone for radical operation, or after. Unfortunately, it is most commonly the latter; and all that you may be able to do will be to ease the end of the patient's life, and simply treat symptoms.

I have here the notes of a fairly typical case of cancer of the cervix, which, in illustration of the subject, I shall read to you.

The patient, aged 56, complained of a blood-stained thick discharge from the vagina, which began shortly before Christmas, 1892. The patient noticed no smell with the discharge. Two months ago, says the report (which was written in March, 1893), she began to suffer gnawing pain in the lower part of her abdomen and back. The patient came to the hospital, and was treated as

an out-patient. Two days afterwards, however, a profuse hæmorrhage occurred, and she was admitted as an in-patient on March 15th. On examination as an out-patient, the cervix had been found to be affected with carcinoma. The left posterior utero-sacral ligament was also involved. The growth was found on examination under an anæsthetic to extend up into the uterus, leaving a small surface at the fundus free. I scraped the patient's cervix, and got away as much as I could of the growth, and then applied Chloride of Zinc.

The cervix of the uterus is usually divided into two parts for purposes of study. This is not a mere arbitrary division, but a very important one, since a different kind of cancer is inclined to affect each part. These are the supra-vaginal cervix, and the infra-vaginal, or portio vaginalis. That is the ordinary anatomical description; but for the purpose of clinical description it is divided in a rather different way, adopted by Dr. John Williams in his Harveian lecture on "Cancer of the Uterus." According to this method the portio vaginalis is considered only as that part in any longitudinal section which lies between a line drawn from the fornix to the external os. The external os means the place where the squamous epithelium covering the outside of the vaginal portion merges into the columnar epithelium, covering the inner surface of the cervix. On the inner surface there are mucous follicles, while on the outer there are no glands. The squamous epithelium and the portio which it covers fit on to the cervix, defined as above, "like a thimble on a finger." It is on the part covered with squamous epithelium that the ordinary epithelioma with bird-nest cells develops, while it is on the other that either columnar or irregular carcinomata develop—carcinomata consisting of acini with cells more or less regularly arranged round them and infiltrating the adjacent tissues, or of acini with irregular epitheliomatous cells.

This division of the cervix is important not only as regards the pathology, but also, as we shall see later, as regards the way in which the cancer spreads. These cancers may take the shape of an excavating ulcer, or they may form nodules. When they affect the *portio* only, they do not alter the shape of the cervix very much; at least, they produce no characteristic alteration; they simply make it larger and more or less irregular. Ulceration on the surface soon

occurs, so that there is simply an irregular-shaped mass which does not present any definite outline. The only well-marked modification in shape is what is called a cauliflower growth. That is a very rare thing indeed, a thing which I have never seen myself except in museums and illustrations in books. As a rule, it grows on a stalk into the vagina, constituting a kind of villous growth which may fill up the vagina. Other growths are called cauliflower growths which are not really such, but are simply the result of the tissue proliferation and general thickening which occur in cancer of the cervix proper. These have no pedicle, and simply consist of a soft spongy mass which somewhat resembles a cauliflower.

In cases where the cervix proper is affected, there may be an excavating cancer. In this case an appearance is presented like that of the crater of a volcano, the excavation being conical in shape, continuous with the cervical canal; its surface is irregular and brittle, extending, it may be, on to the vaginal walls. On the other hand, the growth may be nodular and projecting from the cervix. Or it may form a more or less polypoid mass, though not so regular as an ordinary mucous polypus, which may grow outwards or from the inner side of an excavation. We had a case the other day with several polypoid masses growing from a crater. In addition to these two situations, cancer of the cervix may, in elderly women, begin in mucous polypi which were formerly innocent, but which subsequently develop a malignant character possibly on account of the irritation caused by rubbing against one of the lips of the cervix. It is important to remember this.

As regards the origin of cancers, in the one kind it begins in the squamous epithelium of the part, and not in the connective tissue. All epitheliomata spring from previously existing epithelium. It was formerly supposed that lacerations were the commonest sites for the development of epitheliomata. Lately this has been proved to be erroneous. The microscopic examination of a large number of Dr. Williams's cases showed that cancer had rather a tendency to avoid lacerations than to involve or begin in them; at all events, lacerations are generally free. The operation known as Emmet's operation for the repair of the cervix when it has been split was formerly advocated as a means of preventing the possible irritation that the eversion of the lips

might cause. As, however, it has been shown that cancer does not tend to begin in lacerations that can no longer be urged in its favour.

Cancer of the cervix proper begins in the glands by proliferation of the epithelium; and not on the surface. In this case, too, it does not tend to begin in lacerations any more than in cases of epithelioma of the portio vaginalis. It may, however, begin in the glands beneath the surface of an erosion.

The matter of next importance is the mode of spread. Supposing you have a case of epithelioma of the portio vaginalis, a superficial cancer, as epitheliomata are; the epithelioma does not tend to spread inwards towards the cervix, or deeply into the connective tissue, but superficially. If, for instance, it begin near the external os (it may begin at any part) it tends to spread along the surface, and to involve the vagina. It hardly extends deeper than the sub-mucous tissue; it is entirely a superficial extension, for a very long time, at all events.

In cases of carcinoma of the cervix proper it may extend in three ways. It may extend downwards on to the portio, then it proceeds to spread on to the vaginal wall. The vaginal wall may be reached either by means of the portio or after the portio has been ulcerated away. On the other hand, the cancer may spread, as it most commonly does, into the connective tissue at the side; and on that account carcinoma of the cervix is a particularly unfavourable kind to have to deal with; it so very rapidly spreads into the connective tissue, and, having once done that to any extent, it is quite impossible to stop it by a complete operation, which would remove the whole disease, as you can in the case of so many other organs. Carcinoma of the ovary, for instance, is not common, but when it does occur, if you see the case early enough and remove the ovary, you cure the patient; because the carcinoma remains within the peritoneum covering the ovary, and you may be fairly sure you have eradicated the disease. In carcinoma of the cervix, on the other hand, you cannot.

The third way in which it may spread is into the body of the uterus. This is not very common, but still it does occur, and often enough to justify the question whether the whole uterus ought to be removed at once in every case where carcinoma of the cervix is diagnosed. On this there is not general agreement, but on the Continent this

practice is more widely followed; and by some the whole uterus is removed, even if carcinoma of the portio alone is diagnosed.

In the case of which I gave you the history, there is nothing said about the condition of the cervix in the report. She was first seen by my assistant, but he made no notes at the time. Her left sacro-uterine ligament was involved. On dilating the uterus, it was found that the growth extended up into the uterus, leaving only a small surface at the fundus free. [This was also illustrated by another case in which there was a carcinoma of the cervix which spread into the body.] At a later stage the body of the uterus grew to two or three times larger than normal. Most of the growth on the cervix was bounded by a rim of normal tissue, which encircled it all the way round, except where interrupted by a laceration. No doubt the laceration enabled the carcinoma to get on to the vagina rather earlier than it might otherwise have done, but there is, as I have said, no evidence that a laceration is the site of origin of carcinoma. The vagina thus became involved. It is, therefore, an illustration of all the three directions in which the carcinoma may spread. The sacro-uterine ligaments are not the commonest channels for the spread of carcinoma into the connective tissue around the cervix. The commonest place is on each side of the uterus, that is to say, at the base of the broad ligaments. In cases, however, where you are investigating whether the woman is a fit subject for operation, you must never omit to investigate the condition of the sacro-uterine ligaments, and these may be best examined per rectum.

As regards the kind of patient who has carcinoma, and the influences which render one patient more liable to it than another, I think age is the most important. Patients are more likely to have it between 40 and 50, about the time of the menopause. The frequency decreases as you get back to 24 or 25. Cancer of the cervix has occurred as early as that; and it is not very rare to find it as early as 26 or 27. In other parts of the body cancer does not often occur anything like so early as that; but it must not be imagined that because a patient is not of the age at which cancer usually occurs, that you may leave it out of consideration when the cervix is the seat of mischief.

Heredity has, no doubt, something to do with the tendency to cancer; but cancer of the cervix is so common in women compared with cancer in

other parts that one can hardly say that this cancer occurs oftener in women with a cancerous family history than in those who have not such a history.

Multiparity appears to have no influence whatever in the causation of cancer. It used to be supposed that women who had had a great number of children were especially liable to cancer of the uterus, and that mainly because they had numerous lacerations; but these having been shown to have no effect, that condition as a predisposing cause of cancer falls to the ground. In addition, Dr. Williams's statistics quite bear this out.

Erosions do not produce carcinoma; but they enable it to reach the surface of the portio much more readily than it otherwise would. An erosion is a condition of the portio in which, instead of having its normal covering of squamous epithelium, it has a covering of one layer of columnar or cubical epithelium. There are no glands, normally, in the portio; but where an erosion occurs there are glands, the follicles lying beneath the surface. As it is in these follicles that carcinoma can grow, it will readily invade the portio when there are erosions present. The erosions extend the glandular surface of the cervix beyond its normal limit; but erosions themselves probably have no influence in actually producing cancer.

The classical symptoms of cancer are four. The first is hæmorrhage. *Hæmorrhage* is practically constant; no case, one may say, occurs without some hæmorrhage. In character this hæmorrhage is not profuse. It is not, for instance, like that connected with certain fibroid tumours. It is merely an oozing. Anything like flooding is very rare in this condition, and occurs only in cases where a large or moderately large vessel, such as the artery to the cervix, gets ulcerated through by the growth. As a rule the arteries are thrombosed before the ulceration occurs. This symptom is nearly always the first thing that is noticed. In the three cases of which I have here the reports it is stated of the first that she had been losing a great deal of blood at the monthly periods; of the second that there was blood-stained discharge from the vagina. In the third it is said she complained first of pain in the lower part of the abdomen, the case being one of cancer involving both the cervix and the body of the uterus; but the rule is that hæmorrhage is the first thing to be complained of.

Pain occurs in about half the cases. It is due to involvement of the nerves in the connective tissue around the cervix at the base of the broad

ligament. In the body of the uterus (with which we are not dealing now) the pain before the peritoneum is affected is probably due to the irregular contraction of the uterus set up by the growth. The pain shoots in various directions,—usually down the back of the thighs, especially if the sacral plexus or one of its branches is involved, in which case the pain becomes almost intolerable.

The *discharge* is usually of a watery character, and frequently blood-stained. It is a serous discharge from the surface of the growth, of which it contains small detached particles. It is generally brown, and usually offensive, this offensiveness being almost characteristic. It is due, in early stages of carcinoma, to retention of small quantities of blood in the cervix which decompose, and make the discharge smell. Later on it is due to actual sloughing of the growth. But a patient may go from beginning to end without a foul discharge. We have recently had cases of that kind.

The *wasting* varies very much. It does not occur in any large proportion of cases, and very often not at all till near the end, when the cancerous cachexia develops. You must not think that because a patient is fat she has therefore not got cancer of the cervix.

There are other minor incidental symptoms which do not help in diagnosis, but are often of great importance to the patient, and may cause much discomfort. One is pruritus vulvæ, due to the irritating discharges running over the labia and perinæum. Or there may be disorder of micturition,—frequency, difficulty, or pain. These are mostly due to involvement of the connective tissue between the cervix and the bladder in the growth; they are not due to increasing size of the cervix.

The physical signs depend on the variety of the cancer. I need hardly go over a description of the various shapes. You may be able to pass your finger right up to the internal os, the whole cervix having been ulcerated away. Scattered over the surface there may be nodular projections, or a polypoid mass may project from it. Whatever the shape, the surface itself is extremely brittle. After you have examined a few cases of carcinoma, you will, in ninety-nine per cent. of the cases, have no difficulty in making out the disease by the sense of touch. After the examination, too, there is always some hæmorrhage, and it may be fairly free.

When the cancer has invaded the connective tissue, the growth tends to fix the uterus. It is important to determine whether there is any fixation

of the uterus or not. If present it indicates involvement of the connective tissue about it, and shows the unsuitability of the case for a radical operation. How far the vaginal wall is involved should also be made out. If it is implicated to any great extent, operation is precluded, but when there is only slight involvement, and, without damaging important structures, you can get enough tissue to allow of your dividing the vaginal wall half an inch away from the growth, it is a case in which, in the absence of other contra-indications, you ought to operate. In your examination of the ligaments, the sacro-uterine ligaments should not be forgotten, they may not at first be felt, but they should always be looked for per rectum.

The ulceration in the vagina from the growth in the cervix may cause fistula—the commonest being a vesico-vaginal one. When that occurs the patient's condition is one of extreme misery. She has not only the growth affecting her bladder, causing her pain on micturition and pain at other times, but there is also constant dribbling away of urine, so that her life is specially pitiable.

As regards diagnosis, there are one or two things in connection with which mistake is to be guarded against. The most likely error is to mistake the cancer for an erosion. In most cases it is easy to distinguish between the two, but in very early epithelioma of the portio where there is a flat surface and not much thickening, it is not so easy. In that case the only course is to treat it as an erosion in the ordinary way, watching it carefully lest it spreads. Do not leave it too long, so that it becomes too late to operate. Watch it for a week or two, and find whether the erosion heals, or whether the affected surface spreads, in which case you at once proceed to more radical measures. If it does not spread, go on treating it as an erosion and it will heal up. Time is an important matter in this case.

If a fragment can be obtained for microscopic examination, you may often, but not always, settle the question at once. Carcinoma of the cervix may also be mistaken for chronic hypertrophy, especially with ectropion; the glands in the cervix then becoming enlarged considerably and projecting on the surface, giving the cervix an irregular nodular shape. In cases of chronic inflammatory hypertrophy there is no great hæmorrhage unless there is erosion too. A microscopic examination of a fragment here also will enable you to make out whether it is cancer or hypertrophy.

A sloughing fibroid might also give rise to error; but that, probably, only by its smell, because the relations of the fibroid are usually readily made out. But the possibility of a fibroid or a mucous polypus undergoing cancerous degeneration must be remembered.

Cancer of the body of the uterus you will be able to distinguish from cancer of the cervix, by examining with the finger.

Senile endometritis is another condition in which there is a sero-purulent or purulent or bloody discharge from the uterus. Usually, in that case, there is hardly any pain, and the patient does not get ill, but goes about. The condition may be present for a long time without affecting her health.

The other most likely thing is a retained and decomposing ovum. That may give rise to a foul discharge, and it often causes considerable hæmorrhage. You may feel something presenting at the cervix which momentarily you might mistake for carcinoma.

Prognosis.—In these cases, from when you discover the cancer, if you cannot operate on it, you may calculate the patient has probably not more than eighteen months to live. That is usually the outside limit; but some patients have gone on for five or six years without any appreciable deterioration of health. Most patients die, however, within eighteen months. As a rule, death occurs in one of two ways. It may be caused by exhaustion from the discharges, and from the fact that the patient develops fistula, cystitis, etc., in which case she dies slowly and painfully; on the other hand, it may occur by suppression of urine—and this is the best way for the patient—the suppression being caused by the extension of the carcinoma into the connective tissue and the implication of the ureters; so that the patient dies comatose without pain or disturbance. Of course, she does not have eclampsia; that is to say, she does not die in the condition known as uræmia, with convulsions, but from suppression.

In addition, the patient may die from peritonitis, the growth spreading up into the peritoneal cavity. Or absorption of septic products may occur, and the patient die of septicæmia.

Treatment.—As regards treatment, the cases divide themselves into two classes:—(1) those you can operate on with the hope of eradicating the disease entirely; (2) those in which you can only remove as much of the disease as possible, in

order to make the patient at least more comfortable for the rest of her life, or where you can merely treat symptoms.

Of the indications for deciding on one method or the other, the first is the fixation of the cervix. You have to decide whether the connective tissues around the cervix are involved. Examination per vaginam and per rectum should be made. One of the first things that will strike you in these cases is the absence of mobility of the uterus. After some experience in examining a large number of uteri you will be able to make out whether the cervix is as movable as it ought to be. If it is not, the connective tissue, you will conclude, is involved. If the cervix is freely movable, and the growth has only attacked the lower part of the cervix or portio, you have a very good prospect of being able to get rid of the whole disease. You have also, secondly, to determine the extent to which the vagina has been involved. I have already mentioned the points to be noticed in examination of the vaginal walls.

If you think you can get rid of the whole disease by it you may, in the first place, remove the infra-vaginal cervix, or portio (as it is *ordinarily* understood). That has been done in various ways. If you are quite certain the disease is limited to the tip of the cervix this operation may be done. The *ecraseur* may be used; but that is a method you should prefer not to use, because while you are tightening the *ecraseur* you may get parts of the peritoneum, especially parts of Douglas's pouch, dragged in and cut off. You may also get a wide raw surface, and stenosis of the cervix may result. The best way is to split the cervix into an anterior and a posterior lip, and to make a V-shaped incision in each lip—one limb of this starting from the fornix, to meet at an angle pointing upwards, another from the inside of the cervix. All the portio is thus removed, and the two surfaces of each lip are brought together, and the risk of contraction of the cervix is avoided.

Supposing the cancer involves the cervix, but all is freely movable, then if it extends some distance from the external os, the question arises whether you should remove the whole uterus, or be satisfied with the supra-vaginal amputation. In this latter operation the incision begins at the fornix, and you cut out a wedge-shaped piece from the uterus up to just above the internal os, or still further beyond it. If necessary, instead of making the excised wedge end here you might carry it further

up the uterus and excise as much of the uterine wall as you like; but this makes it more difficult. This is not an easy operation; it is almost as easy to remove the whole uterus, and is probably safer for the patient. Occasionally, too, in cases of carcinoma of the cervix the mucous membrane of the body contains cancerous nodules, or is in a chronic endometritic condition. So if the cancer of the cervix is far advanced, yet not fixing the uterus, it would very likely be better to do a vaginal hysterectomy.

Removal of the portio, removal of the supra-vaginal cervix, and removal of the whole uterus are thus the three operations which may be employed.

In cases where there is no hope of saving the patient's life and preventing recurrence, you may scrape away all the diseased tissue you can remove with a blunt instrument. The instrument I most commonly use is a closed pair of curved scissors, blunt at the end; or a Simon's spoon, not sharp. This will scrape away all the growth till you come to more or less healthy tissue. By doing this you prevent the patient suffering from hæmorrhage for some time. The patient often does not bleed for three or four months after that, and foul discharges also are, in this way, stopped. As a rule, too, she picks up in health and strength. If she has been wasted before, and had indigestion, with general illness from the constant foul-smelling discharge, she gains weight. If you think you cannot get away nearly all the diseased tissue by mechanical means you can apply a solution of Chloride of Zinc to the part you have scraped, the strength used being five drachms to the ounce, that is to say, rather more than half the weight of Chloride of Zinc to the ounce of water. That makes a very powerful caustic indeed. You soak a small pledget of wool in that, and fit it into the part scraped, protecting the vagina at the same time with another piece of wool soaked in a saturated solution of Carbonate of Soda, which neutralises any excess of Chloride of Zinc, and prevents the vagina from being cauterised.

For the smell and foul discharge use an antiseptic douche, a saturated solution of Boracic Acid, or 1-2000 solution of Perchloride of Mercury, regularly night and morning.

For the pain there is no harm in the patient taking as much Opium as she likes. There need be no limit, in reason, to the amount you may

allow her. If she forms an Opium habit it does not matter;—she certainly will form it; but as she has only to live a few months longer, let her have as much as is necessary. If the Opium disagrees with her, making her uncomfortable and confining the bowels, you can substitute injections of Morphia. For constant injections such as will be required, it is well to use a solution with $\frac{1}{10}$ grain of Atropin in it to the $\frac{1}{4}$ grain of Morphia.

A CLINICAL LECTURE

ON

THE DIAGNOSIS AND TREATMENT OF CATARACT IN CHILDREN AND ADULTS.

Delivered at St. Mary's Hospital, November 7, 1893,

By HENRY JULER, F.R.O.S.,

Ophthalmic Surgeon to the Hospital.

GENTLEMEN,—By the term cataract is meant an opacity of the crystalline lens. This may be *partial* and non-progressive, or it may extend to the whole of the lens substance, when the cataract is said to be *complete* or *mature*.

As a matter of fact there ought to be no difficulty in detecting the existence of cataract when it is so advanced as to interfere with vision, yet it frequently happens that children are sent to the hospital by their medical attendants to be treated for "short-sightedness," when the defective vision is due to cataract; adults, on the other hand, are constantly brought before us having been told that they are suffering from cataract when they are, unfortunately, more or less blind from glaucoma, optic nerve atrophy, choroiditis, or some other unmistakable form of eye trouble. This crude ignorance on the part of medical practitioners appears to be taken for granted, or at least tolerated, with regard to diseases of the eye, whilst similar mistakes in almost any other department of medicine would be treated as malpractice. Now that the examining boards are gradually giving more importance to ophthalmic subjects in examinations we may hope that this condition of things will grow smaller by degrees and beautifully less.

As a matter of fact there is nothing easier than the detection of cataract. We have only to dilate

the pupil with a few drops of 2 p.c. solution of Homatropin, place the patient in a semi-darkened room, condense the light from a lamp or candle upon the dilated pupil with a convex lens, and the opacity can be clearly seen both as to its exact position and the extent to which it has involved the structure of the lens. By the direct method of ophthalmoscopic examination the cataract can be seen in a different way, much as we see the negative of a photograph. The light from the mirror is thrown into the area of the pupil, if the cataract is mature there will be no red reflection from the fundus oculi, if the cataract is immature the light will pass through the yet transparent portions of the lens as far as the fundus, whence they will be reflected back again into the observer's eye, and the transparent portions of the lens will appear red, whilst the opaque or semi-opaque portions will appear more or less black. Whilst thus examining by the direct method it is well to turn on a convex lens of strength 20 dioptries behind the sight-hole of the mirror so as to magnify the lens structure, by this means very fine opacities of the lens may often be detected. Such opacities always indicate a progressive tendency and assist us in the prognosis of the case.

When cataract is complicated by old iritic adhesions so that the pupil is bound down and cannot be dilated by the mydriatic employed, of course, it is less easily distinguished. When opacities exist in the vitreous humour these may render the diagnosis of the case more difficult by causing a dull reflection from the fundus, but still the opacity of the lens can be well seen by focal illumination in the manner just described.

Cataracts present themselves to our notice in various stages of advancement. They may occur in persons who are, to all appearances, in perfect health, or they may be found associated with other diseases of the eye, such as cyclitis, iritis, choroiditis, or with general diseases, as syphilis and diabetes. They are frequently caused by injuries of the eye, in which the lens is more or less dislocated or wounded. When a cataract is only partial, the seat of the opacity depends very often upon the disease which has brought it about. Thus we sometimes find an opacity at the anterior pole of the lens, just beneath its capsule; it is well defined, small and non-progressive. It is caused in most cases by ophthalmia neonatorum, in which there has been iritis and probably perforation of the cornea. Opacity is also found at the

posterior pole of the lens, arranged in a radiating manner either within the lens substance or upon the back of its capsule, it is usually associated with choroidal disease, and is, therefore, not a desirable case to operate upon. A third form of cataract consists in a zone or lamella of opacity, situated about midway between the surface and the centre of the lens. This is usually found in children, but it may be so slow in progress as to be unobserved before adult life; it is usually associated with a history of convulsions in infancy, and is probably due to an arrest of nutrition at the time of the child's illness. It is seen by focal illumination and by retinoscopy to be more opaque at its periphery than at its centre, and slight cases may be overlooked altogether owing to the faintness of the opacity. When zonular cataracts are stationary they are smooth and uninterrupted in outline, when progressive they present nodules and protuberances at their circumference, which radiate outwards in the cortex and inwards towards the centre of the lens, so as to form, in time, a complete cataract.

A fourth form of cataract is that in which pyramidal masses of opacity are seen at the periphery of the lens, pointing inwards towards the centre; so long as they do not reach the central or axial portion of the lens they do not cause much inconvenience, and may be disregarded.

The most common form of cataract occurring in adults, and one which gives most visual trouble, is the nuclear form. A cloudy opacity occurs at the nucleus of the lens, and gradually by extension involves its whole substance. The period of this maturation is very variable—from a few months to many years. The colour of such cataracts is usually grey or amber-like, sometimes they have a dark brown colour, and occasionally they are quite black, and consequently difficult to diagnose. When very mature the cortex is apt to undergo fatty degeneration, and to liquify and become milky in appearance, the nucleus floating about within a fluid sac.

In children affected with inherited syphilis, there is sometimes found a cloudy opacity of the whole lens structure, such lenses are usually smaller than normal, their capsules seem to be tough and their suspensory ligaments defective.

It is usually in the chronic form of diabetes which occurs in elderly subjects that we find progressive opacity of both lenses occurring; it is invariably symmetrical, and the cataracts are, as a rule, white.

Cataracts arising from injury occur in every degree of severity, from a mere dot to complete opacity of the lens.

A cataract caused by previous local disease, as irido-cyclitis or choroido-retinitis, is manifested possibly by iritic adhesions or reduced intra-ocular tension, defective or even absence of any perception of light; besides, the lens is frequently in such cases calcareous, and has a dull porcellaneous appearance. The condition of the fundus in the other eye will considerably help in the diagnosis; for example, if disseminated patches of choroidal atrophy are visible in the non-cataractous eye, we can rightly assume such a condition to exist in the other eye, only in a more extensive degree, as the cataract is, in such cases, due to arrest of nutrition.

In order to simplify this important subject it may be well to treat it under two distinct heads, viz., those occurring in children and those in adult life.

1. In children the *zonular* or lamellar variety is that most commonly met with, and though occurring in infancy they do not declare themselves until some time after education has commenced. Children thus affected are often thought to be short-sighted, because they fail to see distant objects distinctly, and in order to get larger images they hold any book they may be reading very close to the eyes. Having dilated the pupils and detected the opacity in the way just indicated, our first care must be to ascertain whether the cataract is stationary or progressive. If stationary, we must note whether there is sufficient space in the non-affected cortical portion of the lens to allow of clear vision. If such be the case, there will be no necessity to disturb the lens at all; it will suffice to make a small iridectomy downwards and slightly inwards, thus making a small peripheral artificial pupil through which the patient will be able to see and will still retain the power of accommodation. In order to be quite sure that an iridectomy will benefit such a case, you have only to cause dilatation of the pupil by Atropine, and, any error of refraction being corrected by proper glasses, to note whether distant vision of Snellen's test types is improved or otherwise.

The patient I now show you is one in whom iridectomy was performed ten years ago. He is now twenty years old; the central portion of the lens, you will observe, is now quite opaque, but the cortex is still clear and free from radiating opacities. He can read No. 18 of Snellen's types

at six metres, and he can read the smallest print without spectacles. He is an employé on the railway, and his vision appears to be as good as ever.

When the distant vision is not improved by atropine mydriasis, you can hardly expect iridectomy to be of service; the opacity is then too near the surface of the lens. The boy I now show you is a case of this kind; there are no radiations of opacity from the periphery of the cataract, but the clear cortical zone of the lens is insufficient for distinct vision. The case must be treated by the needle operation for absorption of the whole lens.

When you find by focal illumination and by the ophthalmoscopic examination that the surface of the cataract is sending out processes of opacity into the clear cortex, this must be taken as a sign that the whole lens will soon become opaque. An iridectomy here would be only of temporary benefit, and if the opacity is such as to interfere seriously with vision, there is no advantage to be gained by waiting; indeed, the younger the child the more quickly will absorption of the whole lens be produced by the needle operation; and, therefore, the sooner this is resorted to, the more quickly will the vision be improved, and the child will have a better chance of educating the eye to its new condition. Indeed, there is nothing to gain but much to lose by postponement of the operation; if left till the age of fifteen years and upwards, the nucleus of the lens has become sufficiently dense to necessitate many sittings before the cataract can be absorbed, and the process often becomes so tedious that the operation of needling has to be supplemented by extraction of the firmer lens substance, either by suction or by means of a curette, through a small incision of the cornea.

2. *Nuclear, or Senile Cataract*, which, as we have seen, is the more common form of opacity occurring in elderly people, commences by gradual failure of vision and an inability to obtain suitable spectacles. One eye is generally worse than the other. In the early stages the patient can often see better when his face is turned away from the light so that the pupil is more dilated. By dilating the pupil and using the focal illumination and the ophthalmoscope in the way just described the diagnosis can be surely established. Care must be taken to distinguish between opacities of the cornea on the one hand, and those of the vitreous body on the other; the former can be easily seen by

focal illumination, whilst the latter can only be seen with the ophthalmoscope. Having found nuclear opacity, we have to ascertain whether it is progressive or stationary, whether an operation is likely to restore the vision, and whether the operation should be performed at once or at a later period. I need hardly tell you that in no case of cataract can we hope to cause absorption of the opacity by therapeutic agents; some operative procedure must, therefore, be resorted to, if any improvement of vision is required. After the age of thirty the structure of the lens is so dense, and its nucleus so large, that the needle operation is no longer effectual; in the first place the number of sittings, and the time required to produce complete absorption of the lens would be too great, and in the second place the swelling of the lens substance caused by stirring it up with a needle, and the greater resistance offered by the sclerotic tunic of the adult eye, combine to set up inflammation and pain as well as glaucomatous tension. Our best plan now is to remove the whole lens at one sitting by extraction from its capsule through a sufficiently large incision at the sclero-corneal junction. For this purpose it is usually advisable to avoid operating until the opacity has extended to the greater part of the lens, otherwise the unaffected cortical substance remains adherent to the capsule; and, although owing to its transparency at the time of the operation it cannot be seen, it afterwards becomes swollen and opaque and gives rise to a secondary cataract which may prove as opaque as the first one, and which will have to be torn through afterwards by one or two cutting needles. When the whole lens has thus become opaque the cataract is said to be mature or "ripe"; the signs of this condition are that no red reflex can be obtained from the choroid by the use of the ophthalmoscope, that there is no darkness in the pupil when light is thrown upon it by focal illumination, and that the patient cannot count fingers when held up at half a metre distance in front of the affected eye. This ripe condition is that most favourable to the complete evacuation of the lens from its capsule; there are, however, many circumstances which render it highly inconvenient, if not altogether impossible, to wait for the complete maturation of a cataract. There may be commencing or equally advanced opacity of the other eye, by which the patient is deprived of all useful vision, and is consequently debarred from following his occupation in life. In such cases the extraction of the immature cataract

at the earliest possible date is imperative. In persons over sixty years of age there is less necessity for waiting than between the ages of forty-five and sixty, as the cortex matter is naturally harder in the older people and will shell out more easily. In cases under sixty it is better to wait until the lens looks fairly opaque and there is not much shadow of the iris as indicated by the darkness of the pupil. If the patient cannot wait, you will facilitate the extraction of the lens and possibly hasten the maturation of the cataract by performing iridectomy upwards as a preliminary operation some six or eight weeks before the extraction of the cataract. Förster and others have advocated the "artificial maturation" of the cataract at the time of the preliminary iridectomy by rubbing the lens through the cornea whilst the anterior chamber is empty, thus disturbing its nutrition by somewhat severe injury. From what I have seen of this method in my own practice as well as in that of others, I am of opinion that it would be safer to extract an immature lens than to injure the eye by violence, which can hardly fail to set up inflammation in its most vital parts, viz., the ciliary body, the iris, and the cornea.

In deciding whether to operate upon a case of mature cataract, it is necessary to consider whether it is what, perhaps, you will allow me to call a "physiological cataract," that is to say, one in which no other morbid changes exist in the other structures of the eye, or one dependent upon, or associated with, some other disease in the eyeball, and which might therefore be called a "pathological cataract." No cataract can be so dense as to prevent perception of light. In testing the perception of light it is important to note, whether the patient acknowledges that he sees the light with the cataractous eye, whether his visual axis is directed towards the light or not, for if the eye wanders and is unable to fix the luminous object it is a sure sign that his yellow spot is not in a healthy condition. If perception of light is perfect it is necessary to test the peripheral part of the retina to see if that is also active. A light is projected into the eye by a mirror from different quarters of his visual field, and if he can accurately point to the position from whence the light is coming we know that his retina is equally active at its periphery, and consequently his visual field is not materially, if at all, contracted. The pupillary reflex should always be tested, and it will be found that an active contraction to light is a strong point in favour of a

healthy retina. A sluggish or inactive pupil will suggest fundus disease, and in many such cases there will also be found no perception of light. The tension of the eyeball should also be recorded. Cataract frequently complicates glaucoma; and, on the other hand, diminished tension will point to disease of the ciliary body and choroid. One small posterior synechia will not prevent your advising an operation, but numerous synechiæ are a barrier to any successful issue. The condition of the other eye, if the fundus is visible, may give valuable information as to the state of the fundus of the cataractous eye.

Before operating it is of paramount importance to have the conjunctival sac thoroughly cleansed, the margins of the lids and the roots of the eyelashes free from any moist or dry secretions and rendered aseptic, and above all, the lachrymal apparatus should always be examined to see that no obstruction exists. These may appear points of little significance, but I can assure you that the success of the operation depends mainly upon the attention to these apparent trifles. If lachrymal obstruction should be present the cataract extraction should be postponed till this septic lachrymal sac has been made aseptic and the passage down the duct opened and rendered patent. The lachrymal sac is a subtle harbour for germs.

The importance of having the instruments, the hands of the operator and his assistants alike rigorously cleansed, is apparent to you all, for a chain is no stronger than its weakest link, and the use of one dirty instrument is quite sufficient to condemn the operation.

It is not necessary for me to describe to you the details of the operation, for you may almost every week see the operation performed in the theatre, and you will learn far better the steps of the operation by seeing it done than by any description I can give you. It briefly consists of an incision at the sclero-corneal junction occupying the upper third of its circumference, the performance of iridectomy if considered desirable, and the extraction of the lens after lacerating its capsule. Whether iridectomy should or should not be performed is a point over which there has been much discussion, and at the present date the opposing views are fairly equally balanced; I will not, however, detain you to-day to give you the reasons in favour of, and the objections to the performance of iridectomy in the operation of cataract extraction.

THERAPEUTICAL NOTES AND FORMULÆ.

Bronchitis.—Dr. J. N. Love says that a preliminary smart purge is most desirable in the treatment of this affection, thus getting rid of irritative matter, ptomaines, etc., from the body; he strongly recommends also the addition of 10 or 15 drops of Turpentine to each dose of the purgative, on the ground that it is one of the best glandular stimulants we possess, and is an ideal drug for bronchitis. This treatment should be followed by Benzoate of Soda in doses of 10 to 20 grains well diluted every hour or two, with this, bandaging the chest firmly is of great use. In children unable to expectorate, an emetic is of the very greatest service. Often, says Dr. Love, when all other remedies have been futile the liberal administration of Quinine will clear up a bronchial catarrh as if by magic, especially if a malarial influence can be even suspected. Turpentine he finds of equal, or even greater use, after the acute stage has passed, with a view to the prevention of a subacute or chronic continuance of the trouble.

(*Med. Rec.*)

Neuralgia.—Exalgin has, in doses of 3 grains, been found very useful in neuralgias of various forms, and in rheumatic pains, by R. v. Weismayr. The dose is repeated, if necessary, as often as four times a day. It is useless against the pain of inflammation or passive congestion.

(*Wiener Klin. Wochensch.*)

For Epilepsy, when Bromides do not effect improvement:—

R. Zinci Oxyd. ... gr.ss
 Extr. Belladonnæ ... gr.ss
 Pulv. Rad. Valerianæ ... gr.xv
 M. Ft. pulv. Mitte pulv. xxx. One powder to be taken three times daily.

NOTE.—On each repetition of the powders increase the Oxide of Zinc, until the dose reaches even as much as ten times the above amount.

Irritable Heart:—

R. Chloralamid ... ʒiv
 Tinct. Bellad. ... ʒij
 Aq. and Syrup ... ad ʒiv
 M. Sig.: ʒj three times a day.

(*Med. Rec.*)

THE CLINICAL JOURNAL.

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A CLINICAL LECTURE ON A CASE OF CEREBRAL SYPHILIS.

Delivered at Guy's Hospital, Jan. 13, 1894,

By W. HALE WHITE, M.D., F.R.O.P.,

Physician to, and Lecturer on Materia Medica, at the Hospital.

THE case of the man before you, Gentlemen, is a very interesting one. You will not often, I think, see a patient from whom you may learn so many important points. I will tell you the facts about him; some of them I am able to demonstrate to you now, others, which have passed away, I cannot. I shall meanwhile give you nothing of his previous ailments, as I wish to show that without knowing anything of them you should be able to diagnose the case.

The patient's friends tell us that last August he attended Dr. Shaw as an out-patient for ptosis, which, under treatment, improved, and that then, and before, and since, he has had considerable mental weakness; and those of us who know him in the ward are well aware that he is mentally defective. The first point then that we have to note about this man as one of his prominent symptoms, and a symptom to be harmonized with the others, is his mental weakness.

The next statement about him in the report is that on the 27th of December last he went to the Crystal Palace; not much is known about what happened there, but he is said to have had a fit, and when he came back his friends noticed that he was weak on the left side of the body. If I were to show you the dynamometer pressure now, you would see this is still the case. He can press to 60 with the right hand, but only to 30 with the left. The arm was especially weak on admission, and has been ever since. We note secondly, then, a fit followed by weakness in the left arm.

The third and fourth points mentioned in the report are that his friends noticed that after the fit his speech was thick and his saliva was dribbling somewhat. When I ask him his age and where he lives, you will hear that even now, although it is three weeks since the onset of his attack, his

speech is thick. He does not at present show much dribbling of the saliva, but he did when he came in.

Passing on with the history we come to his state on admission. At that time it was noticed that he had a vacant expression; he did not seem to be "all there," as the saying goes; his mental condition being decidedly defective — though perhaps, his loss of expression makes that seem more marked than it really is.

On examination of the face, the left side, as you will see, is partially paralyzed. When asked to show his teeth, the angle of the mouth on the left side does not move up well, while on the other it moves easily, and the movement of the left occipito-frontalis is not quite so good as that of the right. The fifth symptom, then, that we have to note, is partial left facial paralysis.

You will notice, too, that when I ask him to put out his tongue, and he has got it steady, that the point is deflected to the right. That means that the genio-hyo-glossus is paralyzed on the right side, and the tongue is pulled to the right by the unaffected muscle on the left side. We note, then, partial right hypoglossal paralysis.

The next symptom mentioned I cannot very well show you as he lies here, and that is, that we think the sterno-mastoid on the right side does not contract quite so strongly as that on the left. Unfortunately, we cannot see his larynx; the epiglottis falls so far down that this is a larynx which it is impossible to see; but, at any rate, the sterno-mastoid being paralyzed shows that he has paralysis of his right spinal accessory. It is very slight; but he says himself that when he uses his right sterno-mastoid to turn his head, he does not do it so easily as when he uses his left. The seventh symptom, therefore, that we have to note is partial right spinal accessory paralysis.

When he came in, too, he had some difficulty of swallowing very well marked indeed, but that has passed off. What that means, of course, is partial paralysis of the branches of his pharyngeal plexus, probably some of the vagal branches, and some of the glosso-pharyngeal; and as the spinal accessory and hypoglossal paralysis is on the right side, this paresis was probably right-sided also.

Other evidence of the paralysis of the spinal

accessory is that the right side of the palate was distinctly lower than the left when he came in. The spinal accessory is now known, you will remember, to supply the palate, although it is not quite certain what course the fibres take to get there. Unilateral paralysis of the palate means paralysis of one spinal accessory. We believe, then, that he has paralysis of the spinal accessory because there is unilateral paralysis of the palate and sterno-mastoid; and I think there is no doubt if we could see the larynx, considering the palate fibres of the spinal accessory are affected, we should find the right cord did not move as well as the left.

With regard to the eye symptoms you will notice as he lies there that the axes of the eyes are obviously divergent, and when I make him look towards the right in the direction of the class, you will see that the right eye is turned out very far indeed, but he cannot turn the left inwards as far as he would like to; he is trying to look at all of you, and he cannot get his left eye inwards far enough to do it. That is to say, the left internal rectus is paralyzed. In the ward, when we asked him to look up we thought that the left superior rectus was paralyzed also. I can see that it is slightly paralyzed now. Thus two branches of his left third nerve are paralyzed.

Another point,—one that does not bear on the diagnosis, but which you can see very well,—is the secondary over-action in the right eye. Why this unparalyzed eye is turned too far when he looks at you is this,—the left internal rectus is paralyzed. He has to send a very strong impulse down to the left internal rectus because it is partially paralyzed; a correspondingly strong impulse is sent to the right external rectus, which turns the right eye too far, because before his illness he was always accustomed to send equally strong impulses to the left internal and right external recti as they always act together. The mind has not yet got accustomed to the paralysis of the left internal rectus, and consequently the right eye goes out too far. That is what is called the "secondary deviation" in the sound eye. It is, of course, of no diagnostic value in this case; it is more a mental phenomenon than anything else. The next symptom we have to note, then, is the partial paralysis of the left third nerve.

You notice that both the pupils are widely dilated. I shall be able, I think, to show you also that they will not contract when a light is

brought near them. You see there is not the slightest alteration when I hold a match right in front of the eyes; the pupils remain widely dilated, and you also see that when we powerfully stimulate the skin of the neck they remain immobile, so that he has paralysis of the iris, for the pupil neither contracts nor dilates. We have tested him in the ward, and you must take my word for it that he has paralysis of accommodation. Therefore we learn that all the internal muscles of the eyes, namely, those of the iris and the ciliary muscle are completely paralyzed. He has, in technical phraseology, complete bilateral internal ophthalmoplegia (the "internal" referring to the muscles inside the eye).

The optic discs are perfectly normal.

We come now to the sensory symptoms. In this connection it is recorded that he has deficiency in his sense of smell, deficiency in his sense of taste, and considerable impairment of sensation on the left side of the body. At present there is impairment in the left side of the face, and in the left arm; when he came in there was impairment in the left leg, but that is no longer present.

With regard to what has occurred during his stay in the hospital, two points are worthy of notice, one is that some days ago when going to the lavatory he had another fit, of which the account is that the whole left side of the body was affected, the face twitching only slightly, and—what is an interesting point—a red flushing of the face occurred, limited to the left side, and lasting for a little while after the patient was put to bed. This, then, was a motor fit accompanied by a vasomotor paralysis of part of the left side of the body.

The other point to which I wish to draw attention is that he has in most respects improved considerably since he came in, the only exception being that the internal ophthalmoplegia has remained stationary.

These, then, are our data, and with these data we have got to make out what is the matter with the man. They are, you may say, a strange muddle of symptoms. The first thing, I am sure, that will strike you in looking at them is their extraordinary variety in position. If we are to have any lesion in the brain to account for symptoms so diverse and occurring on different sides of the body, it must clearly be a multiple lesion. Now a multiple lesion in the brain usually means malignant tumours, or tubercular tumours.

There is not the slightest evidence of either of these. Thus the first line of argument we employ is this: that as the symptoms are so multiple we cannot conceive of any one growth in the brain causing them, and it would even be very exceptional for several growths to cause such a series of symptoms, and they would be growths of a kind that they would show themselves elsewhere in the body. We are driven, therefore, to the conclusion that the cause of these symptoms cannot be inside the brain.

As the lesion cannot be inside the brain, and as anyone can tell from the symptoms that the disease must be somewhere inside the cranial cavity, it stands to reason that you turn to the meninges. Now, if you look at his symptoms you will see that some of them fall into three groups. You have, as a first group, a fit at the Crystal Palace, followed by left arm weakness; a motor fit again the other day on the left side; and some deficiency of sensation. Thus these three symptoms all point to some lesion on the right motor area. In that region you can imagine a lesion which, by its irritation, would affect the opposite side of the body, the arm, the leg, the face, and the sensation. We may, therefore, fairly say that we have got a lesion external to the brain somewhere (roughly speaking) about the right motor area. That will explain the deficient sensation on the left side of the body, because it is a certain fact that lesions of considerable area, in any way impairing the function of the motor area, cause loss of sensation in the paralyzed parts. This is because sensation is represented in the motor area of the cortex; and when the lesion is large, or whenever a large piece of the brain has been cut out of the motor area, and the patient has been carefully tested, it has been invariably found that there is some deficiency of sensation in the paralyzed parts. We may, therefore, say that a lesion outside of the brain over the right motor area will explain why the man has had twice a fit on the left side of the body, and why he still has weakness and deficient sensation there. As he is not left-handed, it is impossible that such a lesion can help to explain his difficulty of speech. Further, those of you who are fresh from your physiology will know that the cortex has some function connected with the vasomotor system on the opposite side of the body, for it is possible, by stimulating the cortex on one side, to affect the vasomotor system on the other. Occasionally, in

man, as in this case, we see flushing on the opposite side of the body following upon an irritative lesion on one side of the cortex. Thus, by locating a lesion over this patient's right motor area, we have explained a good many of his symptoms.

We now pass on to see if there is any other group. There is the right hypoglossal paralysis, the paralysis of the right spinal accessory (shown by the unilateral paralysis of the palate and the sterno-mastoid), and that of the pharyngeal plexus (meaning the vagus and the glosso-pharyngeal). You ask yourself, "Can I locate any lesion outside the brain which will explain these?" Nothing is easier than to imagine, on the right side of the brain, a small lesion which shall implicate the hypoglossal, the spinal accessory, and the glosso-pharyngeal nerves, as they emerge from the side of the medulla. They are all packed so closely together there that any one, knowing nothing of medicine, but fresh from the dissecting room, if he was told that the patient had paralysis of the hypoglossal, of the glosso-pharyngeal and of the vagus, would say, of course, he must have a little lesion at the side of the medulla.

Now we have been able to locate two lesions. Can we fix upon another? Running our eye down the list of symptoms one thing that strikes us is that the patient's left third nerve is paralyzed. We notice also that he has deficient smell and deficient taste. The impressions of taste from the anterior two-thirds of the tongue, ultimately reach the second division of the fifth, passing by the chorda tympani, the facial, the Vidian, and Meckel's ganglion, and those of taste from the posterior third of the tongue also reach the fifth nerve, but their path is not known for certain.

Now, the third and fifth nerves are not far apart at the base of the brain, and we may well suppose a lesion there implicating the left third nerve and both fifth nerves, and perhaps extending far enough forward to explain the loss of smell. Thus we have so far got three groups of symptoms. The first due to a lesion over the right motor area; the second due to a lesion at the side of the medulla; the third due to a lesion about the middle of the base of the brain.

We have come to the conclusion now that the disease is something in the meninges. It is fairly chronic because the man has been ill since August. You will now ask, What are the causes of chronic meningitis? Only two are known—syphilis and

alcohol. Of these alcohol is extremely chronic, and, as far as I know, never causes so many symptoms as this. The drunkard dies from something else long before he gets such a marked group of symptoms as our patient has; so that chronic meningitis due to alcohol gives a totally different clinical picture from that which we have here. It is a slight meningitis, recognized more post mortem than during life. We may, therefore, put alcohol out of the question at once, and diagnose with as much certainty as anything in medicine that the man has syphilitic meningitis—with such certainty, indeed, that you need not ask the patient if he has had syphilis. You may be quite sure, whatever history you get, that a chronic case with diffuse random cerebral symptoms so diffuse that no one lesion will cause them all, is a case of syphilitic meningitis. Having made this diagnosis, we will, in order to convince you of its accuracy, ask the patient whether he has had syphilis; and you hear that it is at once admitted, so that the diagnosis is confirmed. The reason why I did not previously mention that the patient had had syphilis, was that I wished to impress upon you that you ought to be able to diagnose these cases without ever asking the history; so that if you think the question as to syphilis will give the slightest offence you need not ask it. Even if the patient declared it to be absolutely impossible, the patient is wrong and the diagnosis is right.

We have come to the certain conclusion then that the case is one of syphilitic meningitis. What do I mean? What I mean is this,—that dotted about are gummatous deposits in chronically inflamed meninges. These deposits always begin in the pia mater, never in the brain. The “gumma of the brain” is one of the pia mater which has grown inwards, and so implicated the brain. There is, so far as I know, no certain specimen of gumma originally growing from the brain. We believe, then, that this man has gummatous deposits in his pia mater, and we know that in the majority of cases, all round these gummatous deposits there is a local diffuse chronic thickening of the meninges; further, we know that this diffuse chronic meningitis leads to the adhesion of the meninges to the brain. Here is a specimen consisting of a large portion of brain with a gummatous mass on the outside, the membranes all round are thickened and so adherent that they cannot be torn off. Here is a smaller

similar specimen. In it too you can see the gummatous material and the membranes adherent to the cerebral cortex.

We have thus an exact picture of what is going on in the case of this patient,—we have got gummata setting up local meningitis.

You may ask, Can you tell us anything more exact about the pathological appearances in this case? Is the condition more marked in any part of the brain than another? Yes, I can; and this case illustrates the point admirably. When this gummatous meningitis occurs there are three seats which it particularly picks out. The most common seat for gummatous meningitis is at the entrance to the fissure of Sylvius, which is about where we imagined the first group of symptoms had a lesion to cause them. In the pia mater, just about the beginning of the right fissure of Sylvius, some gummatous deposit has in this case taken place, and from it a diffuse meningitis has spread up over the right motor area, and causing our first group of symptoms.

Then you may ask, “I understand that this lesion may so damage the brain as to account for the paralysis, but why has the patient had fits?” The explanation is this. The gummatous deposit has, as we have seen, set up some local meningitis; by the consequent adhesion of the membranes to the cortex they act as an irritant, and the patient consequently suffers from Jacksonian or focal epilepsy, because there is a focal irritant which, from time to time causes motor discharges on the opposite side of the body. The membranes by adhering to the motor area have caused the fits, and when there are no fits there is gradual and slow impairment of function of the motor cells, causing the weakness of the left side of the body which you can yourselves see. There is also, for reasons already given, slight blunting of sensation on the left side of the body from the same cause.

It is a clinical fact that the next most common seat for this gummatous deposit is down the side of the medulla, and we have seen that a lesion here is indicated by the second group of symptoms which this patient shows.

The third most common seat for gummatous meningitis is about the diamond-shaped space between the optic tracts and the crura cerebri. But a lesion here, and spreading a little from this region, is exactly what is wanted to explain the paralysis of the third nerve, and the loss of taste and smell. This man, therefore, is a perfectly

typical case, having his gummatous meningitis in the three most common seats.

Another clinical fact worthy of notice is this: That while many tumours besides gummatous deposits are met with in the motor area, and many growths occur, too, near the diamond-shaped space, growing for instance from the pituitary body, it is rare for any lesion to be found at the side of the medulla except this gummatous meningitis. So that if a patient comes to you with symptoms of paralysis of one side of the palate, of the larynx on the same side, and of the hypoglossal and glosso-pharyngeal on the same side indicating something local about the side of the medulla, the chances are he has got local syphilitic meningitis. You cannot say of either of the other two groups of symptoms that they must necessarily be due to syphilis; but if the only symptoms the patient has point to a lesion at the side of the medulla, you may be almost certain that you are dealing with an early case of syphilitic meningitis in which the deposit has occurred there. It is, therefore, important to recognize this grouping, and you should particularly notice that although the right hypoglossal, glosso-pharyngeal, and spinal accessory nerves were paralyzed, the right facial was not. It is a curious fact that the facial nerve commonly escapes in cases of this local gummatous meningitis at the side of the medulla.

We have thus learned that the characteristic of a syphilitic meningitis is that it is a random scattered disease dotted about the surface of brain, but that it has three particular seats of predilection. So please remember that if you have a patient with a great variety of symptoms which you cannot harmonize with one lesion, the chances are that the man is suffering from cerebral syphilis, as it is roughly called, meaning gummata, often minute, in the pia mater setting up local meningitis around.

Now, let us consider the symptoms we have left out. I can explain these to you. You will notice that I have not hitherto explained why there was dribbling of the saliva. I did not do so, because it is a secondary symptom caused by those we have considered. It was no doubt partially due to the paralysis of the face, partially to the paralysis of the tongue, partly to the paralysis of the palate, and partly to the paralysis of the pharynx—all four combined to cause it; so that although it is very indicative clinically of something about the side of the medulla, and is a well marked symptom of glosso-labio-pharyngeal paralysis, as it

is called, it does not indicate paralysis of any one particular nerve. In this case it is very likely that while some of the difficulty of speech is caused by the patient's mental condition, a good deal is due to the paralysis of those nerves at the side of the medulla.

Before we try to explain the other symptoms, I think we had better see if any syphilitic changes other than meningitis ever occur in the brain, and whether the remaining symptoms can be explained by them.

Supposing a patient has syphilis, you ask me, what cerebral changes can result from that? He may have gummata. That we have discussed. They need not necessarily, although they do generally, set up a local meningitis. Occasionally you get a thickening of the membranes without any gummata, or the gummata may have been so small that by the time you see the case they may have been absorbed, and left nothing but the meningitis.

Another very important result of syphilis we have not yet spoken of is syphilitic arteritis. What may that do? It may produce thrombosis, which causes symptoms that supervene moderately quickly. A man in Stephen Ward, who had ultimately to be transferred to the strong room, had syphilitic arteritis and thrombosis in the middle cerebral just as it distributes itself into the five branches radiating from the fissure of Sylvius. It is common for the arteritis and consequent thrombosis to affect these branches from the middle cerebral. In the case I have mentioned the cortical area affected became so large, that in addition to being hemiplegic the patient became demented and maniacal.

Again the arteritis may produce an aneurism, but that is rare.

Hæmorrhage may also very rarely be produced by arteritis.

In children, syphilitic arteritis may cause chronic induration of the brain, but this is excessively rare.

The last three of these results of syphilis are very, very rare. In the vast majority of cases of cerebral arteritis, if it is going to do any harm at all it will do so by producing thrombosis.

What else can syphilis do in the brain? It can render the patient liable to general paralysis of the insane, and it can cause optic atrophy.

The next effect of syphilis which I have to mention, I am sure you would never guess, namely, that it will sometimes produce in the brain degeneration of the motor cells supplying the muscles

of the eye. They are placed along the floor of the aqueduct of Sylvius and the upper part of the medulla, and the affection which results from their degeneration is called "nuclear palsy." You will say, There are other cranial nerves that have motor nuclei, do not they degenerate? No: for so curiously does degeneration pick out the motor nuclei of the eye muscles, both internal and external, that if you say a patient has nuclear palsy, at once I suppose you to mean paralysis due to degeneration of the nuclei of either the third, the fourth, or the sixth nerves, or of the nuclei supplying the internal muscles of the eye. Nuclear palsy then means degeneration of the nuclei of the oculomotor nerves at the floor of the aqueduct of Sylvius; and if the degeneration affects the nuclei of the external muscles of the eye, the result is external ophthalmoplegia; if it affects the nuclei of the iris and ciliary muscle it will give rise to internal ophthalmoplegia.

The other conditions syphilis can produce in the brain are excessively rare. It may produce a genuine pachymeningitis;—by that I mean inflammation of the dura mater. It may produce a diffuse cerebritis: or, again, it may produce an acute suppurative meningitis. These, however, are so rare as the result of syphilis that they do not enter into practical medicine. No one ought to think of diagnosing them at the bedside; they are mere pathological curiosities. We may, therefore, leave out these exceptional conditions.

We have seen that the man has gummata and meningitis, but this diagnosis does not explain all his symptoms. None of them can be due to arteritis, because the only symptoms it could produce, which are not excessively rare, would come on suddenly, and we have no history of anything very sudden except the fit, and that we believe to have been due to the irritant meningitis. Let us, therefore, again consult our list of the effects of syphilis upon the brain, and we see that it may affect the mind, and we therefore may conclude that our patient's mental deficiency is due to it. Very likely it is allied to general paralysis of the insane, and is perhaps the precursor to it; so that the man has not only got syphilitic meningitis, but he has possibly got incipient general paralysis of the insane, and this in part perhaps explains his thick speech and his impairment of smell and taste. It is noteworthy that unilateral paralysis of the palate is said to be often associated with general paralysis of the insane.

Has he optic atrophy? No.

Then in our list of effects of syphilis on the brain, we come to nuclear degeneration. Has he got that? I certainly think he has. I think he has internal ophthalmoplegia. But you will say to me, "Why do you think that? You say he has paralysis of the left third nerve; will not meningeal deposit on the third nerves explain why the pupils are completely paralyzed, and he cannot accommodate?" No, it will not; and the reasons why it will not, and why we must suppose the specific mischief has produced the degeneration of the nuclear at the floor of the aqueduct of Sylvius are these: In the first place, if the third nerve on the right side were so affected as to render the pupil completely immobile, some of the external muscles of the right eye would be paralyzed. But they are not. There is no paralysis of the external muscles of the right eye at all; so we may conclude that on the right side, at least, meningitis pressing on the third nerve will not explain the internal ophthalmoplegia. And in the second place we believe the patient's internal ophthalmoplegia on both sides is due to nuclear palsy for these reasons:—(1) It is very significant of nuclear palsy that it should be bilateral and equal in degree on the two sides. It so happens, as a clinical fact, that you hardly ever see one eye only affected with paralysis of any of the eye muscles from affection of the nuclei. You could scarcely expect both third nerves to be under the influence of precisely the same amount of pressure from a local meningitis. Therefore, the facts that both eyes are affected, and in equal degree, decide against pressure on the third nerves, and in favour of degeneration of the nuclei. (2) The patient has been treated by specific remedies, and we have been able to get the gummatous meningitis to some extent to clear up, for the symptoms due to it are better, but if the internal ophthalmoplegia is due to actual degeneration of the nuclei of the internal muscles of the eyes we should not expect treatment with Iodide of Potassium and Mercury to improve it. You see the internal ophthalmoplegia is still complete, and that fact is very strong evidence that it is caused by nuclear degeneration. I may add that nuclear palsy is hardly known, except in the subjects of syphilis, and the patient affected with it nearly always has other symptoms of syphilitic affection of his nervous system. You will notice our patient illustrates these two points.

Thus the ultimate diagnosis is that this patient

has gummatous syphilitic meningitis especially marked in the three commonest seats, nuclear degeneration of the nuclei of the internal muscles of the eyes on the floor of the aqueduct of Sylvius, and commencing mental failure.

It will only take two or three minutes to discuss the remaining points of the case.

The Treatment is very important, and the chief thing is to get your patient under treatment early. With all the gummatous material about the vagi, what risks are run! At any time there may occur serious implication of absolutely vital nerves. That is why I give Mercury subcutaneously, so as to get the patient under its influence as rapidly as possible. Formerly it was for the same reason rubbed in the skin, or a Calomel bath was given. Inject once a day deep down into the muscles of the gluteal region one-eighth of a grain of Perchloride of Mercury dissolved in five minims of water. Several hundred injections have been given in my wards during the last three years in cases of syphilis of the nervous system, and only once have we had an abscess, and that was in a man suffering from myelitis, who had trophic sores all over his body, and abscesses occurring in parts of his body where no injections were made. If the syringe is clean there is no reason to fear any bad result.

The next thing about the treatment is the Iodide of Potassium. Give your Iodide in large doses. Begin with five grains thrice a day, at the end of three days make it ten, at the end of a week make it fifteen. This man is getting now fifteen grains three times a day. It is far better for it to be absorbed quickly, therefore quickly work up to a full dose. But do not give it too long, because it is a depressant. Rapidly reach doses of thirty or forty grains a day, and continue it for six weeks. Then give the patient a rest from drugs, and resume the treatment at a later period if you think it necessary.

If the patient can afford to go, it is a good thing to send him away for a sea voyage, and, generally speaking, to put him in the best condition of health possible.

Prognosis. Think what you have to deal with. You cannot possibly absorb the increase of fibrous tissue due to the chronic meningitis. If the gumma is absorbed a scar will remain; just as in a syphilitic liver, the scars of old gummata persist. You cannot, therefore, promise the patient to entirely cure him. You may be lucky, and the gumma may be absorbed to such an extent that no symptoms remain; but even then some remnant of

the disease is left behind, and it may at any time set up symptoms. A few years ago a man was in the hospital for cerebral syphilis; he improved wonderfully under treatment and left us, but a few days after he was seized with a fit and brought here dead. All that was found was the scar of an old gumma on the cerebral cortex. There are, indeed, cases on record in which, after full treatment with Iodide of Potassium a scar was left which produced frequent recurrent fits of Jacksonian epilepsy. In such a case it is justifiable to cut down and excise the scar. Be, therefore, guarded in your prognosis. Remember how much and how little you can do. You may generally tell your patient you can do him a great deal of good, but you must add that you cannot promise to permanently relieve all his symptoms, and that you cannot hope to relieve those of a degenerative nature, such as nuclear palsy. These must remain.

A CLINICAL LECTURE ON ANKYLOSIS OF THE HIP.

Delivered at St. George's Hospital,

By T. PICKERING PICK, F.R.C.S.,

Surgeon to the Hospital.

I HAVE, at the present time, under my care, in Princess Ward, a patient on whom I performed Adams' operation, that is to say, subcutaneous division of the neck of the thigh bone, for ankylosis of the hip-joint, some six weeks ago. I do not think I can do better than devote the time at our disposal this afternoon than by taking this case as our text, and drawing your attention to the subject of ankylosis of the hip, and the means that we have for the relief of this affection in certain cases. I need not detain you very long by a lengthy recital of the notes of the case upstairs.

Ada McE., æt. 24, was admitted under my care on August 9th, 1893. In October, 1892, she had a miscarriage, due to an accident, at two months. This was followed by a very severe illness, with great fever and rigors. It was accompanied by very severe and intense pain in the left hip, both knees, and to a less extent in the ankles, with some swelling of these joints. The fever and pain continued very severe for two

months, and then abated; but she was confined to her bed until April of last year, a period of over six months. When she attempted to get about, she found she was unable to stand, owing to the stiffness of the joints which had been affected, and the distortion of the left leg. She stated that she had always enjoyed good health up to the time of her miscarriage, and her family history was good.

When admitted, she was found to be a delicate, pale-looking woman. The left thigh was adducted and flexed, and immovably fixed in this position. No amount of force which I could employ, under an anæsthetic, caused the bone to yield in the slightest degree. The amount of adduction was so great, that the left limb, when she was lying on her back with the pelvis straight, crossed the junction of the middle with the lower third of the sound thigh. And the flexion was so considerable, that the affected thigh could not be brought down into a horizontal plane with the trunk, even though very great lordosis was produced. There was also fibrous ankylosis of both knee-joints in a straight position, and to a less extent of both ankle-joints. There was, however, some amount of movement, or at all events "yielding" in all these articulations. The patient was otherwise healthy. There was no disease in the lungs, and the urine was natural.

During my absence from town, Mr. Sheild placed the patient under the influence of Ether, and broke down some adhesions in the knee-joints. After this, massage and passive motion was steadily persevered with for two months, and under this treatment a fair amount of movement was obtained in both knee-joints and also in the ankles. The left hip-joint, however, remained *in statu quo*, and though several attempts were made to overcome the adhesions, not the slightest amount of movement could be obtained in this articulation. I therefore proceeded on November 2nd to perform Adams' operation. Introducing the long, narrow-bladed knife, which is used in this proceeding, midway between the anterior superior spinous process of the ilium and the upper border of the great trochanter, I carried it down to the front of the neck of the thigh bone. I then passed an Adams' saw along the track thus formed, and sawed through the greater thickness of the neck of the bone. The rest was easily broken, and the limb brought down to its normal extended position. The small wound was closed with a single horsehair suture, and dressed, and the limbs placed in a Bryant's splint

with extension, by means of an accumulator, to the left leg. The case went on most satisfactorily, the temperature was never registered above 99° F., and on November 24th the wound was dressed and found to be healed. The stitch was removed. On December 6th she was put up in a plaster of Paris splint, and she will go home in the course of a day or two.

I think there can be no doubt, from the history of this case, that the patient suffered from septic synovitis, with resulting ankylosis, and that this ankylosis was probably fibrous and not bony, but, at all events, it was so firm that no force, which we felt justified in employing, was sufficient to cause it to yield, and therefore it was necessary to resort to a cutting operation. I shall have something more to say on this subject directly, but it will be more convenient to defer what I have to say until we have considered the different varieties of ankylosis which may occur in a joint.

Stiffening of a joint may vary from slight limitation of movement to complete rigidity and absence of all motion. In the former case, where the movements are only limited, but not altogether lost, the limitation is due to fibrous adhesions which may be either within the joint or outside. In the latter, where there is absence of all movement, the rigidity may be due to bony ankylosis or to dense fibrous bands between the two articular surfaces, so strong that no force which we can employ will cause them to yield.

I am inclined to think that ankylosis, as a result of hip-joint disease, occurs most frequently in those cases where the disease has commenced in the synovial membrane, and the ankylosis may be either fibrous or bony. Here are two specimens from our museum of complete bony ankylosis of the hip—synostosis, as it is called, in which, I think, there can be no doubt that the disease was originally a synovitis, for you can distinctly trace the outline of the head of the femur, and there has been no destruction of bone tissue, with a slight exception, to which I will allude in a minute. Undoubtedly, bony ankylosis does occur in that more common form of morbus coxæ which begins in the cancellous tissue in the neighbourhood of the epiphyseal cartilage; but in these cases there is much greater destruction of bone, and it is important that you should differentiate between these two conditions, as we shall see when we come to speak of the treatment.

Let me sketch briefly the pathology of a case

where the disease begins in the synovial membrane. There is, as you will see, first, a destructive process, and then, secondly, a reparative process.

First, there is inflammation, with its attendant exudation, and a gradual conversion of the synovial membrane into granulation tissue. This tissue creeps over the cartilages and fills the joint, and the layers covering the two surfaces, coming in contact, coalesce, and thus the cavity of the joint becomes obliterated. It gradually destroys and replaces the cartilage and articular lamella of the bone, and thus we have two raw bony surfaces joined by a mass of granulation tissue. Now, this tissue generally undergoes fatty degeneration from insufficient blood supply, and, later on, softening, and becomes converted into a flocculent curdy pus, and thus a chronic abscess is formed, which gradually reaches the surface, and is opened or bursts, and thus the greater mass of this newly-formed tissue is got rid of. But the whole of the tissue does not perish in this way: that portion of it which is nearest the living healthy structures, that is to say, nearest the vascular supply, does not die, and in this a process of repair commences. In this a developmental process takes place, and it becomes gradually converted into fibrous tissue, in which lime salts are deposited, and finally it becomes converted into bone, and thus we get complete bony union, or synostosis, between the two exposed articular surfaces of the joint. It is quite possible that all these changes might occur without the formation of pus; and just as in some cases of Potts' disease of the spine we get destruction of the bodies of the vertebræ and so-called angular curvature, without the formation of matter—*caries sicca*, as we term it—so it is possible that in these cases of joint disease we may get the granulation tissue destroying the synovial membrane, the capsular and other ligaments, the cartilages and the articular lamella of bone, and then, instead of perishing from inanition, becoming converted into fibrous tissue and then bone, and so forming a complete bony ankylosis.

When the disease begins in the bone we have the same changes, but they are more extensive. The head of the bone is infiltrated with inflammatory exudation, and becomes replaced by granulation tissue and altogether destroyed, or some portion of it may die before it has undergone this change, and may remain as a necrosed fragment. Here, therefore, there is a much larger amount of granulation tissue, and therefore it is more prone

to undergo degenerative changes, because the greater mass of it is further removed from any vascular supply. So the granulation tissue which has replaced the bone becomes converted into pus, and is discharged through sinuses, together with any necrotic fragments which have not been destroyed, until the whole is gone, except a layer in immediate contact with healthy living tissue. This obtains the necessary nutrient supply from these tissues, and undergoes the same reparative process as in the other form. It becomes converted into fibrous tissue and then into bone, and so osseous ankylosis results. You will see that here there is more destruction of bone, and consequently more deformity. If you examine such a case after the process is complete, you will note the alteration in the relative position of the great trochanter to the bony landmarks of the innominate bone, the shortening of the limb, and the scars of the sinuses and the puckering of the skin; and in this way you will come to a diagnosis on a point which may have great bearing on your treatment. Of course, too much stress must not be placed on the presence of scars, because as we have seen in those cases of the synovial form of the disease, suppuration may have taken place, and there may be the scars of sinuses; but there will not be the same amount of deformity, and no appreciable alteration in the position of the great trochanter.

Then, secondly, we may have fibrous ankylosis. And in these cases you need have no destructive process going on, though in many instances, no doubt, you do have destructive changes in a minor degree. For this ankylosis may be simply due to a synovitis with the formation of fibrous bands within the joint, as a result of the inflammation; or it may be due to thickening and induration of the fibrous capsule and other ligaments. But we may have also a destructive process, as in the bony ankylosis. In these cases, however, there is not the same amount of destruction of tissue; the cartilages and synovial membrane being only in part removed, and their place supplied by granulation tissue, which in its turn is converted into fibrous tissue, but not into bone.

The amount of limitation of movement which this fibrous ankylosis produces, may vary very much. In some cases there may be only one or two slight bands, which prevent extreme flexion or extension; and again, on the other hand, the fibrous union may be so strong that it is impossible,

even under an anæsthetic, to obtain the slightest movement between the two bones. These latter cases amount practically to the same thing as cases of bony ankylosis, since no force which we are justified in using is capable of stretching or rupturing the union. Generally, however, where the union is fibrous, a slight amount of "give" or yielding may be obtained by firm pressure under an anæsthetic, and it is important to ascertain this point, for the treatment of the two cases differs very materially.

And now we proceed to discuss the treatment. What is to be done for these cases? The first point which we have to consider is whether ankylosis has taken place in a true or faulty position. If in a true position, probably in the majority of cases it is best to leave things alone, unless the amount of adhesion is slight. If the union is bony, clearly this is the only thing which we can do. The patient has got a sound limb on which progression can be accomplished without pain or discomfort, and without much perceptible alteration in gait, by a sort of rotatory movement or twist of the pelvis, and nothing that the surgeon can do can remedy this.

There are, however, certain inconveniences attending this condition. I remember, many years ago, seeing the late Sir Prescott Hewett cut a man for stone, who had an ankylosed left hip-joint, and he experienced very great difficulty in reaching the bladder, and extracting the stone. Probably, now-a-days, under these circumstances, we should select the supra-pubic route. You can understand also that in women, during parturition, an ankylosed hip in a straight position is a source of difficulty both to the patient herself and also to her attendants; and again, the sitting position is a difficult one to assume. But these are minor evils and have to be borne, for I know no means, where the union is bony, by which they may be overcome.

Further than this, I do not think that in those cases where there is firm fibrous union of the hip in a favourable position, that any good results will be obtained by an attempt to break down the adhesions, and an endeavour to restore motion by passive movements. In the first place, the attempt will probably fail. It is exceedingly difficult to fix the pelvis, and so get a firm point from which to work. And even if you succeed in breaking down the adhesions, they probably reform again in spite of all your attempts at motion. And, moreover, serious damage may be done from the

violence necessary to overcome the adhesions. When, however, the adhesions between the articular surfaces are slight, and especially in those cases where they are extra-articular, the perfect mobility of the joint may be obtained by long-continued persevering movement, combined with massage and sometimes subcutaneous division of opposing bands.

Far different is the case where the ankylosis has taken place in a faulty position, as is not unfrequently the case.

You are all, of course, familiar with the fact that in the later stages of hip-joint disease the limb has a tendency to assume a position of adduction and flexion. This is probably to be explained by the distribution of the obturator nerve, but this is a point into which I cannot enter at the present time, nor is it necessary that I should do so. It is sufficient for our purpose to-day that I should mention the fact, and tell you that when ankylosis occurs in a faulty position, as a result of hip-joint disease, you will generally find that it is in this position of adduction and flexion that the ankylosis has taken place.

No doubt this condition is sometimes due to want of attention on the part of the medical attendant, and I should not be doing my duty if I did not warn you in treating these cases to bear in mind that stiffening of the joint may be a very probable result, and that you must, therefore, devote all your energies to maintaining the limb in a straight and perfect position.

But I do not think the medical attendant is always to be blamed. Sometimes it is impossible to prevent it. There are many little circumstances which may occur in the conduct of these protracted cases which prevent the surgeon maintaining the limb in the position he desires, and faulty ankylosis is the result. Now consider what will be the effect on a patient with his hip-joint ankylosed and his limb in a position of flexion and adduction. Directly he attempts to stand, in order to produce parallelism of the femora, he has to rotate the pelvis forwards through a horizontal axis drawn through the two acetabular cavities, so as to overcome the flexion, and this, of course, produces an anterior curving of the lumbar spine, or lordosis. In the same way, in order to overcome the adduction, he has to raise the pelvis on the affected side, thus producing apparent shortening of the affected leg, and in order to preserve his equilibrium he has to curve his spine in the opposite direction, and

so produces a lateral curvature, which, if the ankylosis is not remedied may become permanent.

Thus a patient with an ankylosed hip in a faulty position becomes exceedingly deformed and misshapen. There are, again, other evils, especially in females, connected with menstruation and parturition, which arise out of this condition.

It is, therefore, absolutely necessary that something should be done to remedy it, and what ought to be done depends upon whether the union is osseous, or such firm fibrous union as not to allow of any motion between the two bones, or whether the union is fibrous, and of such a nature as to be capable of being stretched or ruptured.

In the former class of cases there are two operations which can be done for this condition. For I do not do more than allude to Sayre's operation, which I have never performed, and which, in my opinion, does not possess any advantages commensurate with its disadvantages. His operation has a twofold object, rectification of the position of the limb, and the establishment of a false joint. It consists in cutting down upon and exposing the end of the femur, and making a section of the bone above the trochanter minor, so as to retain the attachment of the ilio-psoas tendon to the shaft of the bone, for the purposes of flexion. The section is made in a somewhat peculiar manner by means of a chain saw, a semicircular piece of bone, with its convexity upwards, being removed, and the upper end of the lower fragment being afterwards rounded off, so as to imitate a ball and socket joint, and to allow of movement without any chance of the one bone slipping off the other. The two operations to which I more especially refer are known as Adams' operation and Gant's operation, from the names of the respective surgeons by whom they were first proposed and practised.

Adams' operation consists in subcutaneous division of the *neck* of the femur. The operation is performed as follows: A knife, shaped like a tenotome, but attached to the handle by a long narrow shank, is introduced midway between the anterior superior spinous process of the ilium and the upper and anterior angle of the great trochanter, and is carried straight down to the neck of the femur. The knife is then withdrawn and a strong narrow-bladed saw, with an extent of cutting surface of about an inch and a half, set in a long narrow shank like the knife, is passed down to the bone, which is sawn through from before backwards.

There is no great difficulty about the operation, the only thing to be borne in mind is that the saw must be carried at right angles to the axis of the neck of the bone, and the altered direction of the shaft of the femur must be remembered. In cases like those to which I have alluded, where the ankylosis has taken place with the limb in a position of adduction and flexion, it will be generally found that the line of the saw should be about parallel to Poupert's ligament, and if the bone is sawn in this direction it will be found to divide it without any great difficulty. After the section of the bone is completed a pad of antiseptic gauze is to be placed over the wound and bandaged there, and the limb brought down into the straight position. Occasionally, before this can be done, it will be found necessary to subcutaneously divide the tendons of long contracted muscles, such as the adductor longus or rectus, but as far as my experience goes there is never any difficulty in getting the limb into a good position. The best form of apparatus to fix the limb after the operation is, I think, undoubtedly a Bryant's double long splint. By means of this splint the leg can be got absolutely straight, whilst at the same time extension can be made by means of the elastic accumulator.

Gant's operation consists in subcutaneous division of the femur *below the trochanter*. It is performed as follows: The patient lying on his back, and the front of the thigh grasped with the left hand, with the thumb placed rather below the outer side of the femur, a tenotomy knife, similar to that used in Adams' operation, is introduced by the side of the thumb and carried down to and across the front of the femur. The thumb is still kept in position and the knife withdrawn and an Adams' saw introduced along the track over the femur, which is then to be sawn through. As soon as this is completed the saw is withdrawn and the wound dressed, and the limb put up in the straight position on a Bryant's double splint or other suitable apparatus. In both operations, if the proceedings have been carried out with care and cleanliness, the wound heals without suppuration, and osseous union takes place in about eight to ten weeks. Of the two operations I decidedly give preference to the first, as producing the best results, but there are certain cases in which the division of the *neck* of the femur is not applicable.

First, in those cases of tuberculous disease of the hip-joint of which I have spoken, where the

disease has commenced in the bone, and where there has been destruction of the head, and probably the greater part of the neck of the femur, so that only the smallest amount of bone connects the shaft and great trochanter with the pelvis, and, secondly, in those cases where there has been a deposit of new bone around the joint, producing great thickening around the neck of the femur. In these two cases Gant's operation is the one to be performed, in the first class of cases because there is no femoral neck to divide, and in the second on account of the great mass of bone requiring division.

Let me now illustrate what I have said by a narration of the cases which have come under my own care, in which I have performed one or other of these operations.

Case 1. Louise E., æt. 15, came under my care in the Victoria Hospital for Children, in 1886. As a child she had suffered from hip-joint disease, and had been treated for many months by extension in another hospital, and eventually recovered, and went home. Gradually her leg appeared to shorten, and she became bent and distorted, and could not stand upright without producing great deformity of the spine.

When admitted, the thigh was flexed on the pelvis to nearly a right angle. When she stood on both feet on the ground, the trunk became much twisted, and there was great lordosis, producing very considerable deformity; when the affected limb was placed on a stool in front of her, the spine became quite straight. The pelvis was not tilted to one side, and consequently there was little or no adduction of the limb. No motion whatever could be obtained in the hip-joint by forcible movements under chloroform. Here probably the ankylosis was fibrous. She had left the hospital where she was treated for the hip disease with a straight limb, but ankylosed; the union, however, not being sufficiently firm and strong, had gradually given way under the weight of the body, and flexion had taken place. This view of the case explains the absence of adduction which was noted.

As I have already told you, these cases of very firm fibrous ankylosis are to be treated in the same way as osseous ankylosis, and therefore I performed subcutaneous section of the neck of her femur. The limb at once came down into a good position. She was placed on a Bryant's splint, and made a good recovery, with a straight limb.

Case 2. Miss X. Y. Z., æt. 6, came under my care in 1880 with disease of the hip-joint, which had already, probably, existed for some time, but had been disregarded; the pain and limping from which she had suffered being looked upon as a matter of no great importance,—a too common history in this class of cases. There were the ordinary signs of hip-joint disease, with which I need not trouble you. She was kept lying on her back for some time with extension by means of a weight and pulley, and appeared to be doing well, free movement returning in the joint. The parents were anxious to leave London, and she was therefore fitted with a moulded leather splint, and instructions were given that she should be kept lying. Some time afterwards, as I subsequently learned, a recurrence of the disease took place, from what cause I was unable to ascertain. This was accompanied by very intense pain in the hip, and the child was for some time, I am told, very ill, but eventually the pain subsided, and the patient recovered. During this time she was treated with a long splint.

I saw her again in the autumn of 1886. She was then a miserably deformed little object. The affected thigh was adducted and flexed, and fixed in this position. When lying on her back, with her pelvis straight, the thigh was thrown across the opposite thigh, and it was necessary to raise it considerably to overcome the lordosis which otherwise existed. When standing with both feet on the ground, there was very considerable lateral curvature, and it was necessary to raise the foot about twelve inches to render the spine straight, and do away with the bowing forward of the lumbar portion.

Under the influence of chloroform not the slightest movement could be obtained in the joint; the two bones were immovably connected, and moved as one piece.

I had the advantage of Mr. Adams' opinion in this case, and he agreed with me as to its being a very suitable one for the performance of the operation of subcutaneous section of the neck of the femur.

I accordingly performed this operation early in 1887. The thigh at once came into good position, and was put up in a Bryant's splint. The wound healed by first intention, and at the end of six weeks, when there appeared to be already very good union, she was done up in an immovable apparatus of millboard.

I had the pleasure of seeing this patient last summer. She is now a well-formed, perfectly erect girl, and manages the slight limp due to her ankylosed hip remarkably well, so that there is a scarcely perceptible limp, and one which I think would hardly be perceived by a casual observer.

Case 3. I first saw Miss A.B.C. in Jan., 1883, she was then suffering from commencing hip trouble of two weeks standing. Two of her sisters had died of tuberculous meningitis, and she was a delicate child who had had two attacks of pneumonia and also otitis media and perforation of the membrana tympani, so that she was essentially of a tuberculous constitution. I prescribed the usual treatment, and saw her once or twice, when matters seemed to be going on well. I then lost sight of her, and did not see her again till October, 1886, when I found that she had an ankylosed hip in a very faulty position. There had been no suppuration, but the hip was firmly fixed, and not the slightest movement could be obtained. There was a very marked lateral curvature, and, indeed, it was for this that she was brought to see me. The amount of flexion of the thigh was not great, for when she was lying flat on her back, with the affected thigh resting on the bed, there was not any considerable amount of lordosis. Mr. Adams also saw this case with me, and we determined to recommend operation. This was done, and I divided the neck of her thigh-bone subcutaneously. She had no drawback afterwards and the wound healed at once. The lateral curvature was very much improved by the operation, but I fear that the proceeding had been undertaken rather too late, after the bones had become fixed in their new position and probably altered in shape, for there is still a slight lateral curvature, which I fear is permanent.

Case 4 was one in which I performed Gant's operation, and will therefore serve to illustrate the class of cases in which I think this operation is to be preferred. Agnes R., æt. 9, was admitted into the Victoria Hospital for Children on October 14th, 1892. It appears that when she was five years old she was attacked with hip-joint disease which went on to the formation of abscesses which discharged for many months, and then healed up. When she was admitted the right thigh was found to be ankylosed to the pelvis in a position of flexion and adduction. In order to overcome the lordosis it was necessary to raise the foot thirteen inches from the ground. As she was a very small child for her

age, the gait was most peculiar. There were numerous scars of old sinuses about the hip, and the great trochanter could only with great difficulty be made out, as it was buried deeply close to the dorsum of the bone.

Clearly this was a case which was not applicable for division of the neck of the femur, for the neck was gone and there was none to divide. I therefore divided her thigh-bone just below the trochanters, by the operation which I have described to you as suggested by Mr. Gant. The child made a good recovery. The wound was found to be healed upon removing the first dressing, and the bone united in the course of a couple of months. The leg is now quite straight, but shorter than the other. This, however, is remedied by a cork sole and heel to her boot, and she now walks in a very different way to what she did when she was admitted.

This represents the whole of my experience of these two operations—four cases of Adams' operation and one of Gant's. In all the cases the wound healed without trouble, and all were very materially benefited by the proceeding.

And now one word in conclusion about those cases of fibrous ankylosis in a faulty position—where the bands of union can be made to yield, and where there is some movement in the joint. In these cases the limb may be restored to a useful position by forcibly rupturing the adhesions under an anæsthetic, supplemented by the subcutaneous division of contracted tendons or dense bands of fascia, if any can be felt. But before I speak of this treatment, let me say a word or two on those borderland cases, in which it is difficult to say to what extent the joint is stiffened. Occasionally it happens that the rigidity of the muscles around the joint is so great in old standing cases of fibrous ankylosis, and the capsular and other ligaments are so thickened that at first sight we may be inclined to believe that we have to deal with an osseous or very firm fibrous ankylosis, which nothing but a cutting operation will remedy. A decision on this point should never be come to, therefore, without an examination under an anæsthetic, when if the fibrous bands are capable of being torn, a certain amount of yielding will be felt. In the hip-joint we are not likely to mistake an osseous ankylosis for a fibrous one, but in some joints, notably the ankle, this is likely to occur. On account of the mobility of the neighbouring bones and joints, it may easily happen that the surgeon may be induced

to think that some movement is taking place in the affected joint. There is another point which I may mention as a means of diagnosis between fibrous and bony ankylosis. In fibrous ankylosis any forcible attempt to move the joint is accompanied by pain and reflex contraction of the muscles, whereas, in bony ankylosis there is, as a rule, no pain, unless indeed very considerable and possibly unjustifiable violence is used.

Let us suppose, now, that we have a case of ankylosis of the hip, which we believe to be fibrous; in which under an anæsthetic we feel a certain amount of yielding, and in which, therefore, we determine to restore a more useful position of the limb by forcible rupture. The first point to ascertain is, how much of this stiffening is due to rigid muscles? It is astonishing sometimes to feel the firmness with which bones are held together at a joint from this cause, and how rapidly they yield to continuous extension. Some of you may remember a patient of mine who was some little time ago in Burton Ward. A girl, æt. 19, who had old hip disease, and who came into the hospital with her thigh rigidly fixed on the pelvis at a right angle, and how by keeping up extension by means of a pulley and weight of fourteen pounds we brought the leg down to a straight position in a fortnight or three weeks. I am always, therefore, in the habit, before I attempt to forcibly rupture adhesions in the hip joint, to see what can be done to remedy the deformity by continuous extension. You will remember we tried it in the patient upstairs, whose case I alluded to at the commencement of my lecture. But here we did not derive the slightest benefit from the treatment; the deformity was due to causes which could not be remedied by this means.

When you have done all you can in this way, you proceed to an attempt to place the limb into such a position as will be most useful to the patient; and this, of course, in the hip, is the extended one. The patient is placed under an anæsthetic, and the pelvis having been fixed by an assistant, the limb is forcibly and suddenly flexed, and in this way the adhesions may be broken down, and will be heard to give way with an audible snap or crack.

You will note that I said this was to be done by a movement of flexion. Forcible extension in these cases is to be avoided as much as possible, for fear of endangering the artery. I have seen in another joint, the knee-joint, the popliteal artery

ruptured by an attempt to break down adhesions by a movement of extension.

After the limb has been got down into its proper position it should be kept at perfect rest by means of a long splint, and evaporating lotions applied. Sometimes the pain and inflammation which follows this proceeding is very considerable, and the patient is unable to bear any passive movement for a long time: under these circumstances probably adhesions reform, and the joint becomes permanently stiff. In spite of this, however, you have accomplished a great deal, and have converted a comparatively useless member into a very useful one. In other cases the amount of inflammation which follows this forcible rupture of the adhesions is very trivial, and in these cases you may employ passive motion, massage, and hot douches at an early period of the case, and in this way very materially increase the mobility of the joint or completely restore it to its proper functions.

A CASE OF DOUBLE OVARIOTOMY

**In which a Pair of large Wells's Forceps
was left on one Pedicle for Fifty Hours.**

Recovery.

BY

ARTHUR H. N. LEWERS, M.D. Lond.,

Obstetric Physician to the London Hospital.

M.C., AGED 18, single, was sent up to me by Dr. Thomson of Clapton on account of having an abdominal tumour, and admitted into the London Hospital under my care on June 12th, 1891.

The patient had only noticed the swelling about three weeks before admission, and she complained of nothing but the increase in the size of her stomach.

The catamenia appeared when she was 13, and from that time till she was 14 had recurred regularly every four weeks; but since then, a period of four years, she had "seen nothing" at all.

Present state.—On examination, the abdomen was found to be occupied by a large tumour, rising out of the pelvis, and extending upwards to the epigastrium. The surface of the tumour was irregular. Its consistence was different in different regions: for while in the umbilical region it was distinctly elastic, in other parts the tumour was

hard, especially in the left iliac region, where it was almost as hard as bone. On the whole the characters were those of a solid tumour. Dulness extended upwards to within one inch of the tip of the xiphisternum, which was easily felt.

Locally the hymen was found to be bisepatus, a band of tissue dividing the vaginal orifice into two parts. The sound, which could not be passed till the cervix was fixed by means of a tenaculum, entered $3\frac{1}{2}$ inches with the curve forwards and to the right. A hard mass, subsequently found to be a portion of the larger tumour, was felt occupying the left posterior quarter of the pelvis.

Operation, June 29th, 1891.—The abdomen was opened in the usual way, and the surface of the tumour exposed. It was found impossible to appreciably diminish the size of the tumour by tapping, though three attempts were made with a medium-sized ovariectomy trocar. I therefore extended the incision for a distance of four inches above the umbilicus, and removed the tumour entire. It proved to be a tumour of the left ovary. The pedicle was an extremely broad one and very thick, especially for the inner half of it. It was also a very short one in the direction from the uterus to the tumour. The pedicle was transfixed and tied in the usual way, and a ligature tied round the whole pedicle as an additional precaution; the tumour was then cut away. A simple unilocular ovarian tumour, about the size of a child's head, was found on the right side. This also was removed in the ordinary way, and the sutures for closing the wound were inserted ready for tying. It was then found that free bleeding was taking place from the inner half of the left pedicle, the part which has already been referred to as being especially thick and fleshy, and where, owing to the shortness of the pedicle, the ligatures were very close to the uterus. Another transfixing ligature was passed, and tied as tightly as possible. After a few moments, however, it became slack, and bleeding recommenced. I therefore clamped the inner half of the pedicle with a pair of Wells's large pressure forceps, and determined to leave it on. The peritoneum was then washed out with weak Iodine water ($\frac{3ij}{Tr. Iodi}$ to Oj water), and a glass drainage tube having been passed down into Douglas's pouch, the wound closed as usual. The pair of large forceps left on was of course projecting nearly at right angles from the lower end of the wound. Carbolic gauze was packed in large

quantity at the sides of the forceps, so as to take off the pressure as much as possible. Subsequently the case was dressed very frequently, at first every four hours, and afterwards every six hours. The procedure adopted on each occasion was to draw up any fluid from the glass tube by means of a syringe, and afterwards to allow a little weak Iodine water to run in, this again being sucked out with the syringe.

During the time the forceps was left on the patient vomited several times, but not more frequently than is common during the first two days after abdominal operations. The pulse during this time was very frequent, at midnight on the second day reaching 150, and the afternoon of the third day, when the forceps was taken off, being 144. After the morning of the fifth day, when it was 120, it rapidly declined to about 100.

The tongue during the first two days was coated with a brownish-yellow fur, and was dry. The patient during the same time was very restless. There was not, however, any considerable distension of the abdomen.

The forceps was removed at 1 p.m. on July 1st, fifty hours after the operation. No bleeding occurred after removing the forceps. On the whole the symptoms during the time the forceps had been on, though severe enough to make one anxious, were not more severe than often occur in the first two days after abdominal sections that end favourably. The highest temperature, which was only 100.8, occurred at midnight on the second day. After the fourth day the temperature was never above normal. The glass tube was taken out on July 9th. My reason for having it in so long being that it seemed likely that the portion of the pedicle that had been grasped by the forceps must slough. There was no reason, however, for thinking it did so.

The tumour weighed 11 lb. 5 oz., and on section was found to be, for practical purposes, a solid tumour. There were a large number of cysts scattered throughout it, but none larger than a cherry, and separated from each other by firm solid tissue.

A week after the operation the wound measured $7\frac{1}{2}$ in. The patient made an uninterrupted recovery, and went home on August 10th, 1891. I have seen her several times since and she is quite well. She has never menstruated since the operation, and has become very stout.

Remarks.—So far I have not been able to find

mention of a case of ovariectomy where pressure forceps have been left on as described in this case, though the method has often been employed in hysterectomy for fibroids. The question, of course, occurred to me at the time as to any alternative; it was quite clear that no ligature could be trusted, and the only other plan by which the danger of hæmorrhage could have been averted seemed to me to be supravaginal hysterectomy. Remembering how one leaves pressure forceps on the broad ligaments with impunity after extirpating the uterus through the vagina, there seemed to be no obvious reason why the danger should be greater if pressure forceps were left on the broad ligament in an abdominal operation, and the result in this case appears to bear out this impression. Péan has in several cases of abdominal hysterectomy for fibroids been obliged to leave a number of pressure forceps sticking up from the wound, and with good results.

This is the only case that I have met with where there has been any difficulty in securing the pedicle of an ovarian tumour in the usual way by means of the ligature, and no doubt it is only very rarely that any means other than the ligature are necessary for the purpose. Still, the case just described shows that the ligature cannot be universally relied on, and it is well to bear in mind that, in cases where it fails, it is possible to secure the pedicle with pressure forceps with a good prospect of recovery.

THERAPEUTICAL NOTES AND FORMULÆ.

Trional in Insomnia.—Dr. Bryer has lately had very favourable experience of the use of Trional in cases of insomnia. He says that in all cases, almost without exception, sleepiness is produced and rapidly followed by dreamless refreshing sleep. The duration of the sleep depends in some degree on the dose; in correctly proportioned doses no bad after-effects were experienced. If pain is present it may be combined with Opium or Morphia. As regards dose, Dr. Bryer states that men require from 10 to 15 grains more than women, and that a commencing trial dose is 17 to 25 grains for a man and 12 to 15 for a woman; these doses require to be much increased (doubled even) if any acute mental excitement is present, such as mania and hallucinations.

(*Arch. f. Psychiatrie.*)

Acute Rheumatism.—Professor Bourget lays down the following propositions regarding external application in acute rheumatism.

1. Salicylic Acid is rapidly absorbed through the skin. Young skins have a greater power of absorption than those of more elderly patients, and the skins of blondes greater than those of brunettes.

2. The vehicle used to dissolve the Salicylic Acid has great influence on the absorptive power of the skin. Fats increase the power, while Vaseline and Glycerine diminish or almost extinguish it.

3. The treatment of the joint affection by the following ointment is much to be recommended.

Acid. Salicyl.	1 part
Lanolin	"
Ol. Terebinth	"
Adeps	10 parts

M. Ft. unguent. Sig.: To be freely smeared on without rubbing, and then the joint covered with flannel.

4. This is quite useless in the gonorrhœal form, and much less useful in forms other than the acute articular.

5. For muscular and neuritic forms the following powder is often followed by splendid results.

Phenacetin	gr. iv
Salol	gr. iv
Caffeine	gr. ½

M. Ft. pulv. mitte tales xij. Sig.: j to be taken three or four times a day.

(*Rev. Med.*)

Dressing for Burns.—Bismuth Subnitrate mixed with *boiled* water sufficient to form a paste or cream, which is then thickly spread on the burnt parts, and the dressing not touched except to put on more paste as may be necessary to fill in cracks. Blisters must be opened with a prick, and the paste applied over the dead skin.

(*Deutsch. Med. Wochenschr.*)

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THE CLINICAL JOURNAL.

WEDNESDAY, FEBRUARY 14, 1894.

A LECTURE ON FACIAL PARALYSIS.

Delivered at the National Hospital for the Paralysed and Epileptic, by

W. R. GOWERS, M.D., F.R.S.

GENTLEMEN,—Complete paralysis of the right side of the face, in a child of seven, who presents no other symptoms,—that is the problem before us. You have heard the questions asked, and the answers given by the child's mother, and that we found no evidence of a cause; you heard the mother give a negative answer to every inquiry,—there had been no blow, no exposure to cold, no discharge from the ear. Those are the three chief causal facts to be sought in every case. Even pain in the region of the nerve was denied. But one other question was asked that should never be omitted in such a case: Has the child suffered from earache? The mother then remembered the fact that had been forgotten, remembered it with surprise, so little had she thought it connected with the affection of the face.

You see the symptoms which the child now presents, and you have heard the story of the onset.* You see that the face is symmetrical at rest, but in movement all symmetry is lost in the distortion produced by the limitation to one side of all the familiar movements that the will can cause, or by which emotion is expressed. There is no movement, voluntary or emotional, in any part of the face.

Paralysis of all the muscles supplied by the facial nerve, on one side only, and without other symptoms, always means disease of the nerve trunk. Practically, moreover, if it occurs without obvious disease or injury near the nerve after it emerges, it means disease of the nerve during its passage through the bone. These two facts should be fixed in your mind, and the reasons for the conclusion should also be clearly understood. Never learn a diagnostic rule, indeed, never accept any general assertion, without also endeavouring to ascertain on what the rule depends, or the assertion

rests. Unreasoned conclusions are the bane of students. Without the facts to make them cohere with our previous knowledge, such conclusions will soon slip from the memory, or if they do happen to be retained, it is only by dint of pressure which alters their form, or drives them into some place to which they do not properly belong. They are remembered wrongly, and do you more harm than good. But fix the assertions by their evidence, the rules by their reasons, and not only do they remain, but they take root, and they become part of your real knowledge and a source of increasing power.

You are no doubt familiar with the distinction between the two chief forms of paralysis of facial muscles,—the general palsy of all parts which is often, as here, absolute, and the form in which only the lower part of the face is involved, which is never absolute. The first, as you know, means disease of the nerve, or of the nucleus from which the nerve comes, while the other (so common as part of hemiplegia) generally means disease of the cerebral hemisphere, and always means disease of the path between the cortex and the nucleus, or disease of the cortex itself. Hence, the latter is sometimes called cerebral facial palsy, but this is not an accurate term, because this form may result from disease in the crus, or in the upper part of the pons. A more accurate name, therefore, and one by no means inconvenient, is *supra-nuclear* palsy. On the other hand, the general palsy of the face may be either *nuclear* or *infra-nuclear*. It is often called "peripheral," but here again we have a word which it is wise to avoid. If it does not mislead, it is liable to distort a student's first conceptions. Such distortion involves a waste of mental effort, since it has to be rectified. The "periphery" properly means the distal extremity; "peripheral palsy" should be due to disease of nerve-endings—such paralysis as many poisons produce, not rarely, in other parts. But the word is not used in that sense in reference to the face. It is used to mean disease of the fibres anywhere in the course of the facial nerve, and even palsy that is produced by a lesion of the nucleus within the pons—that is of the central origin of the nerve, is sometimes spoken of as "peripheral."

This point,—the use of the word "peripheral" in connection with palsy of the face, may excuse a

* From a report by Mr. H. Caiger.

digression for a moment, to note an illustration of that oscillation of opinion that always attends advancing knowledge. The truth of yesterday may be untrue to-day and true again to-morrow. The grounds of an induction may become insecure when we know the facts more thoroughly, and yet, again, new facts may re-establish that which seemed exploded. When many of those who now teach, first studied medicine, they were taught that the nerve endings are the seat of the lesion in the commonest forms of facial palsy—that which follows exposure of the side of the head to cold. The cold, acting on the surface, was assumed to act on the nerve endings in the muscles of the face and thus to cause the paralysis. The same explanation was given of other palsies following an exposure to cold, such as the local atrophic palsy of children, "infantile paralysis." This pathology was purely hypothetical, no evidence of it had been ascertained, and there was no demonstrable analogy that could be produced to support it. Indeed, it now seems to us strange that the opinion should have been accepted so generally, since a little consideration will show how great a difficulty there must always have been in adjusting it to the facts. Why should cold act upon the extremities of one nerve, upon all of them up to the middle line, and no further, not a single muscle or fibre on the other side. Why should the effect of cold acting on the surface be absolutely limited to the endings of one nerve? This difficulty does not seem ever to have been recognized. When increasing precision of observation, and a wider comparison of facts, suggested irresistibly that such a paralysis must be due to a process acting on the fibres where all are near together, or on the contiguous structures from which they come, and when it was discovered that the latter was the true pathology of infantile palsy, the idea that disease of nerve endings was a cause of paralysis disappeared. All such palsies were ascribed to disease of the centre or nerve trunk. Again, new facts came, and have compelled a partial reversion to the old opinion, that a primary affection of the terminal parts of nerves is a frequent cause of palsy. But it is chiefly in the limbs that we find evidence of this. We have, indeed, returned to this opinion in some cases of paralysis of the face, but we have not returned to it as an explanation of the palsy that is limited to one side of the face, and for which it was once considered the adequate explanation. It is very unlikely, however the patho-

logical pendulum may swing under the impulses of new discoveries, that we shall ever find reason to think that such palsy as you see before you in this child, is ever the result of disease of the nerve endings. We shall presently note the significance of this.

Let us return to the two main forms and their distinctions—the infra-nuclear palsy which is often complete, and the supra-nuclear palsy that is never complete. You perhaps know the hypothesis by which the difference is explained; it is indeed somewhat more than a mere hypothesis. In proportion as muscles act together on two sides, the muscle on each side is represented in both hemispheres of the brain; that is, either hemisphere can act on the muscles of both sides. Hence, in disease of one hemisphere of the cortical centre, or the path from that centre, the muscles escape in proportion to their bilateral association. When I say they escape, I ought to say they either escape, or they suffer only at first. They may be affected for a few days, but the initial weakness soon passes away, at least in great measure. Apparently the structural arrangements by which the hemisphere acts on those muscles on its own side are only slightly in habitual use, but their functional capacity quickly increases. This point, however, is one that we must consider another day; it would lead us too far from our present subject. It is the *complete* palsy—complete in range—of which an instance is before us, with which we are now concerned.

We can carry the diagnosis beyond that of the exclusion of supra-nuclear disease. Paralysis of all parts of the face, if it exists alone, and is on one side only, means disease of the nerve after it has left the cranial cavity. Theoretically it does not; the whole nerve *might* be affected alone within the skull. But, as a matter of experience, it is not. The statement I have made is true in fact. Further, if there is no indication of a cause acting on the nerve after it has emerged from the canal in which it passes through the bone; such a paralysis means disease of the nerve within this canal.

This conclusion is, I need not say, of the utmost practical importance. In a large number of cases of facial palsy—in the majority indeed—it takes us at once to the seat of the disease. But to be able to use such a diagnostic rule you must know its reasons. Without a knowledge of the reasons few such rules are useful, because you cannot feel

so sure of their validity as you must feel if you are to employ them with the confidence that is essential. You cannot feel sure that there are no exceptions, unless you realize why there cannot be exceptions. The facts on which this rule is based are these:—First, there are two portions of the nerve which cannot suffer alone on one side only: these parts are the beginning of the fibres and their terminations—the cells from which they spring, and the structure in which they end within the muscle. These parts are so situated that all of those of one nerve cannot be affected on one side and alone. The nerve cells occupy a considerable area of the pons, and those for the muscles of the eyelids are some distance from those for the lips. The cells are adjacent to other important structures. Hence, the whole of the nucleus cannot be damaged by one lesion, so as to affect all parts of the face without other structures suffering, and suffering in such a manner as to give rise to obtrusive symptoms. Moreover, this is true not only of the nucleus but also of the fibres proceeding from it during their passage through the pons. All the fibres of the nerve are never damaged within the pons without adjacent structures being also damaged so as to cause conspicuous symptoms. Thus the isolation of the paralysis of the face, the affection of the whole of one nerve and of that only, excludes an organic lesion within the pons.

The nerve cells may suffer, and may have their function abolished apart from what is commonly termed an organic lesion. They may cease to act in consequence of a degenerative process, or in consequence of the influence of a toxic agent. This is true, also, as I have already intimated to you, of the other extremity of nerve fibres—their termination within the muscles. These are now known to be occasionally the seat of degenerative processes, and they are also known to be the parts influenced by certain poisons. But such agents do not act only on the nerve endings of one side. The reason for this is important, and, as I just now said, must be specially noted. It is a general law of extreme importance. The same structures on the two sides possess the same pathological susceptibilities. They suffer together in degenerative processes, and they suffer together under toxic influences, whether these be what we commonly term poisons or whether they are only seen in what we call diseases. Thus both sides of the face may be paralyzed throughout as part of diphtheritic paralysis, and as part of multiple

neuritis, due to some toxic agency,—but one side of the face never suffers alone. This is the point to which I adverted in speaking of the theory that cold acts on the extremity of the fibres. Our present knowledge shows that it could not affect the terminations of all the nerves on one side and none of those on the other. Such a cause must, if it acts on the extremities of the nerves, cause bilateral paralysis. As a fact, it does so. Remember, then, in all parts of the nervous system, and in relation to all cases of the character I have mentioned—those due to degenerative processes and to toxic influences—the effect is bilateral; and a complete palsy of any part on one side practically excludes such general causes.

The law which underlies these facts, therefore, may be expressed thus: A palsy which is directly due to a *general* cause is bilateral; conversely, a bilateral palsy indicates a general constitutional influence. A unilateral palsy is due to a local cause; it is not the direct result of a general process. I use the word “direct” result in order to be strictly accurate. The meaning of the restriction is this. The direct effects of general causes are due to their action on the nerve elements themselves; but general states sometimes cause local effects by acting on the vascular system, or they may predispose all parts to suffer from an influence which acts only on one. Moreover, a unilateral palsy means a process which begins outside the nerve elements and affects them secondarily. That is a most important general law, applicable to all parts of the nervous system—most important to remember in all circumstances and in all localities.

Next, the limitation of the palsy excludes disease of the nerve within the skull. After it has left the pons, a process outside the nerve fibres cannot reach a considerable degree and affect the facial nerve alone. The auditory nerve is contiguous to it from the surface of the pons to the bottom of the internal auditory meatus, and a process external to the facial nerve, sufficiently severe to paralyze it completely and in all its parts, must affect the auditory nerve as well; often it affects other nerves that are in the neighbourhood. On the other hand, a cause acting on the facial nerve after it has passed through the skull will be conspicuous; it is always an injury or a considerable local inflammation, and, in either case, is obvious. Hence, therefore, you can now under-

stand the foundation of the important diagnostic rule which I laid down before: that a complete unilateral palsy of the face, without other symptoms, must mean disease of the nerve as it passes through the bone.

The diagnostic problem is thus narrowed in regard to the seat of the lesion, and this facilitates the process of diagnosis even more than may at first sight appear. We cannot, indeed, carry further the process of exact localization, except that, if the short length of nerve which intervenes between the origin from it of the vidian above and the chorda tympani below, is affected, taste is lost on the side of the tongue in front. But this is of little real value, because a morbid process may begin at one spot and may spread through a considerable extent of the nerve. The state of the palate gives us no localizing information. Paralysis of the palate is never produced by disease of the facial nerve. The belief that it is so is one of those curious pathological myths which have arisen from the misinterpretation of what may be termed normal abnormalities, if you will forgive the expression—deviations from perfect symmetry, which have no significance for us except in so far as they may lead us into error.

But the special aid in diagnosis which we derive from this restriction of a morbid process to the part of the nerve which lies in the narrow winding canal in which it passes through the temporal bone, is due to the aid it gives in the next step in diagnosis—the step which is of chief practical importance, the *nature* of the lesion of the nerve. In this part of its course the nerve suffers from only three morbid processes, and of these three one can be excluded without difficulty. The processes are:—(1) primary inflammation of the sheath and interstitial tissue, which is the cause by which the most common form is produced, that which is due to cold; (2) the spread to the nerve of inflammation due to ear-disease; (3) its compression or destruction in consequence of a growth in the bone. It is the last of these which may safely be put on one side unless there are other obtrusive indications of a growth, because it is a very rare cause, and not to be thought of unless such indications exist.

The diagnosis between the two forms of inflammation, that which is communicated from the ear, and that which is primary (the so-called "rheumatic" form), is seldom difficult. They differ in the period of life at which they chiefly occur;

although each may be met with at any age, ear-disease is a rare cause except in childhood, while primary neuritis is seldom met with till childhood is over. Moreover, the mischief in the ear which spreads to the nerve is generally considerable in degree and in duration. In most cases there is actual disease of the bone, and it is the progress of the caries that brings the associated inflammation into such proximity to the nerve that this becomes affected. In such disease there is almost invariably perforation of the tympanum and a constant discharge from the ear. If there is no history of discharge, disease of the ear is not likely to be the cause. But this rule, true of facial paralysis in general, is not invariably true of it. Exceptions are met with, and this case is an illustration of the fact. We often hear vaguely of exceptions which "prove" or "test" the rule. The rules to which this saying is applicable are in most cases general rules; they are laws that are true of the majority of instances which come under them; if these are separated and scrutinized, it will be found that the exceptions occur under special conditions, and that these special conditions have been ignored in formulating the general statement. We have to be chiefly guided by the majority of cases, but we should recognize the existence of exceptions, and know when they may occur, that we may search for their indications if we have any reason to suspect an exception to our rule. If we attempt always to give weight to them, such weight as we give to the majority, we shall be in constant uncertainty. Indeed, it is not unlikely that we may come to the conclusion, as a distinguished scientific man remarked to me of the impression left on him by one of the most famous teachers of his early days, that "no sane man could make a diagnosis."

The exception to the rule that obtrusive signs of caries long precede the facial paralysis which results from otitis, depends upon the anatomical conditions of the ear. This has not indeed been actually proved, but we know that exceptional conditions often exist; they explain that which would be otherwise inexplicable, and which nothing else explains. In some cases, fortunately not common, the facial nerve is separated from the tympanic cavity by a layer of bone so thin that inflammation can readily pass from the cavity to the nerve sheath. In such, bone disease is not necessary for the extension of inflammation to the nerve.

You can now understand why I asked so carefully about the earache. It is not enough to ask if there has been discharge. Discharge generally means disease of bone: disease of bone is the common cause of secondary facial paralysis, but the nerve is sometimes affected by extension when there is no bone disease. This case seems to be of that character, and hence I am anxious to impress its facts upon you. It is an illustration of one of the two chief simple sources of error in diagnosis, not seeing the common, not knowing the rare. I say simple causes as opposed to the more complex sources of error in reasoning.

This is an instance of the rare; it is an example of the occurrence of facial neuritis by extension from the middle ear, without bone disease. It is exceptional, because it must depend on exceptional conditions. A "passage" of inflammation implies a way for it to pass. Normally, there is no way free enough for the passage of simple inflammation. But simple inflammation may spread from the lining membrane of the tympanum to the nerve, when the layer of bone which separates the tympanic cavity and the nerve is thin. Indeed not only may it be thin, it may be even actually deficient. Inflammation not only may, but, if considerable, must then spread to the nerve. Vessels also pass through the bone, and by these an intense inflammation may pass; but it is doubtful whether a simple catarrhal inflammation, such as this child seems to have had, would do so, if the conditions were normal.

One other point, and that of great importance, the case also illustrates. The earache of children is almost always due to inflammation of the middle ear. Most of you know its character from the recollections of childhood, for few children escape some attack, and the peculiarity of the pain impresses itself on the memory. It is one of the maladies that may be called "domestic diseases"; few mothers dream of sending for medical assistance for what they are pleased to call "simple earache." But between the earache which lasts a few hours and then passes off, and the earache which is the prelude to a suppurative inflammation, there is every gradation. There seems to be no difference in the character of the pain.

It is important to remember this. It is one of the facts that should be part of the education of the mother—one of the many facts that might, with great advantage, replace the rubbish that has drifted into the maternal mind: by no means

harmless in the minds of the many persons who "get on much better without a doctor." I lately saw a child with infantile palsy of one leg. This leg had suddenly become powerless. The mother—with no excuse of poverty or station—consulted her female friends, and, resting content with their assurance that it was "only the teething," and that "the leg would soon get all right again," allowed five whole months of utter immobility to pass before she thought it necessary to ask her doctor to look at the child's leg. And many cases of suppuration in the ear and of disease of the bone are due to neglect of the warning of simple earache.

In the case of children who are liable to earache, great care should be taken to guard against exposure of this part of the head to cold, and especially to cold east winds; and still greater care should be taken to get rid of an attack as soon as possible. How seldom are the lessons learned that are, nevertheless, familiar in proverbial form, such as that embodied in the adage about the "stitch in time." We could wish the statement were literally true, and that the absence of early treatment, which so often would cut short a grave disease, could be made up for by the amount represented by the multiplier of the proverb.

If you take all the nerves of the body, and consider the frequency in which they are so diseased as to cause symptoms, I think the fifth, the sciatic, the ocular, and the facial nerve would be their order. The reason for the frequency of facial paralysis is not yet entirely understood. We can understand, however, why it is so obtrusive when it does occur. It is manifested with peculiar readiness, because outward swelling is prevented by the rigid walls of the canal. Hence the inflammatory effusion compresses the nerve fibres, and at once interrupts the conduction of the motor impulses, quickly causing inability to move the facial muscles. But this, although it explains the fact that even a slight inflammation causes considerable palsy, leaves its actual frequency still mysterious. Paralysis of this nerve from cold is certainly more common than we should anticipate, considering that other nerves are not less exposed. Perhaps the cause is to be found in some conditions of the circulation within the canal, in consequence of which a congestion that would otherwise be transient and harmless, leads to undue stasis, and becomes an actual inflammation with all its grave results.

The more remote causes of the primary neuritis are obscure. It is occasionally associated with the diathesis that causes fibrous and muscular rheumatism, and which is probably not far removed from that which causes or results from gout. I have twice known a patient to have facial neuritis at one time, and, at another, an analogous rheumatic inflammation of all the nerves at the back of the orbit. Remember, too, that what we call fibrous rheumatism is probably also not very different from an inflammation. Certain it is that this still mysterious muscular rheumatism may become inflammation. Many cases of sciatic neuritis, certain and severe, arise by the traceable extension, along the fasciæ to the sciatic notch, of a primary lumbago.

We will not attempt to ascertain, in this child, the electrical irritability of the muscles. We know what condition we should find. No complete paralysis of a motor nerve continues for a month without the nerve fibres degenerating below the lesion, and there is always loss of all irritability of the nerve trunk, loss of the faradic irritability of the muscles, and increased irritability of the muscular fibres to voltaism. We should learn nothing by the examination, and you have frequent opportunities for observing the facts. Without doing good to ourselves, we may do harm to the patient. The paralyzed muscles will need electrical stimulation, and in the case of children it is necessary to be extremely careful in the application of electricity. If you so use it as to cause pain the child will be frightened, and will dread each therapeutical application. Once thoroughly frightened, a child seldom loses the dread, and no child can endure a frequent distressing emotion without harm. If care is taken, all that is needed, or almost all, can be achieved without the production of any of this injurious alarm. But to secure this result the first application must be so feeble that no new sensation is produced. Indeed, it is well, the first time, not to allow the current to be strong enough to be felt. Then, if the current is gradually increased in strength in successive applications, after a few days a strength may be used that will make the muscles contract visibly without eliciting a tear. It is surprising how strong a current children will bear if this plan is adopted. For the same reason you should never use the faradic "current" in the case of young children. Move the hammer with your fingers instead of permitting it to oscillate automatically. The less frequent momentary currents will cause a muscle to contract with merely a pecu-

liar shock-like sensation, devoid of pain. If you use the repeatedly recurring shocks, produced by the automatic interruption, a painful stimulation of the sensory fibres is produced, even with a lower current than will cause the muscles to contract. The difference, in the case of children, is of great importance, for the reason I have mentioned. For the same reason, also, if you find it impossible to cause visible contraction of the muscles without producing distress, be content with a little weaker current. I cannot understand the prevalence of the notion that in any therapeutic procedure, with drugs, electricity, or any other agency, no good is done unless a certain physiological effect is produced. Surely the production of a manifest effect is only a question of degree. If a certain strength of current cause a visible contraction of a muscle, it does so by making a large number of the fibres contract with sufficient energy for the effect to be seen. Are we to assume that a current just below this strength causes no contraction because we cannot see the whole muscle shorten? There must be contraction of the fibres before we reach that degree which causes visible movement, and it is surely impossible that this stimulation can be without the influence on the nutrition of the fibres, and on the maintenance of their irritability, which the stronger current has. On the contrary, the consideration of the fact suggests distinctly that such a weaker current only needs to be continued for a longer time to do all that the stronger current can do. Remember, all that electricity can do in such cases is that which I have mentioned. We have no reason to believe that it has any influence on the nerve fibres; it does seem to keep the muscles in a better condition for the nerve impulses to act upon them when conduction is restored, and to enable them to respond better to the motor influences when these can again reach them. Hence, in all cases in which the paralysis is complete, or considerable, for more than two or three weeks, it is desirable to employ electricity.

Moreover, we do not here need an electrical examination to assist in forming a prognosis. This is, it is true, one of the most important services electricity renders in such cases. It is surprising to those who cannot read its language, that of two cases of facial palsy alike complete and of the same duration, say two weeks, an electrical examination should enable a positive statement to be made that one case will be well in a fortnight, and that another will endure for

months and will never completely pass away. And yet it is so. But we have other aids in prognosis. We can often draw conclusions of great value from the simple consideration of the conditions under which a malady developed, and, when a process in one place is due to extension from the same process elsewhere, the character of the primary symptoms is sometimes of significance. Here we have the fact that the paralysis resulted from an inflammation which was transient. The pain in the ear lasted only a few hours. Doubtless the process may have lasted longer, since when effusion and swelling occur, the pain of inflammation often lessens, but the duration of pain which is due to inflammation in the ear is of some value as a guide to the duration, and, therefore, to the intensity, of the process. Thus judged, the process here was brief, and, being brief, was also slight; for duration and degree are proportioned in acute inflammation. From this we may conclude that the inflammation of the nerve was probably only such a brief moderate process as would be limited to the sheath to which it first extended. The effects have already lasted a month, but before long some signs of improvement may be expected to occur. Over and above this, we have also the general law that cases of facial paralysis from ear disease, on the whole, run a more favourable course than cases of facial paralysis from the primary neuritis. The reason for the average slighter severity of the affection is, perhaps, to be found in a more limited extent of the inflammation. It is reasonable to assume that an inflammation which is communicated will not affect so considerable an extent of the nerve as one which is due to a primary process.

In all such cases of facial paralysis, the treatment must be twofold. Besides that suited to any recognizable constitutional state (which I assume you do not need to be told) we have to treat the primary disease of the ear, and we have to treat its consequence. In the case of ear disease, this must first engage our attention, but the measures needful do not come within my own province to describe in detail. The most important, however, is unquestionably to afford a free exit to any pus which may have accumulated in the cavity of the tympanum, and to guard against any obstruction of such exit, including that which is produced by non-absorbent cotton wool. Absorbent cotton wool changed two, three, or six

times a day, according to the amount of discharge, is safe. The importance of cleansing by antiseptic washes you will have already learned. For the secondary inflammation of the nerve sheath we can do nothing directly, except by the application of counter-irritation. This is, indeed, the chief local measure in all cases, whether produced by cold or produced by extension. A blister should be applied over the mastoid process, and should be repeated as soon as the skin will permit. Never put a blister in front of the ear, over the place of exit of the nerve. You cannot blister without producing a little subcutaneous cellulitis, and I have known a trifling cellulitis in this situation to be a cause of facial paralysis—the inflammation reaching the nerve sheath, and spreading along it into the fallopian canal. In all recent cases, moreover, hot fomentations applied over the ear and its neighbourhood, constitute a very useful measure. They should be used for one quarter of every hour during the first day. We do not precisely know how hot fomentations act, but it is certain that no measure has so potent an influence on inflammation in its earliest stage. Probably the stimulation of the sensory nerves and the heat combined, cause alternate contraction and dilatation of the vessels, which lessens the blood stasis. Perhaps, moreover, the nerve stimulation has some even more direct influence on the process of inflammation. But whatever the explanation, the fact is certain.

The last element in the treatment is the application of electricity. This is only needed when the nerve fibres undergo degeneration. As long as the nerve retains any degree of excitability, and as long as the muscles contract when the induced or faradic current is applied to them, electricity is not needed. Its use is to keep up the nutrition of the muscles and to keep up their excitability. We have conclusive proof that it does the latter; we have no proof that it does the former, because we do not find that the muscles to which it is applied waste less or waste more slowly than those to which it is not applied. But it is certain that functional excitability cannot be maintained without nutrition being also influenced. You know the kind of electricity that should be applied. This is one of the elementary facts which every student learns or should learn as his first acquisition in the therapeutics of the nervous system. You must apply the kind of electricity to which the muscles respond, and this, as we have seen, is the

voltaic current. You apply it in such a manner that it is interrupted, not frequently as the induced current is, but slowly, as by *stroking* down the muscle with one electrode, the positive or the negative, to whichever the muscle is most sensitive. It is not always the same, even when the nerves are degenerated. We interrupt slowly because, as far as we can judge, the element in the form of electricity which causes the difference in the reaction is one of time. Muscular tissue is much lower in the scale of excitable tissues than is nerve tissue, and seems to be unable to respond to an electric current unless this has a duration considerably exceeding the very small fraction of a second which elapses before the automatic interruption stops the current of the induction coil.

A CLINICAL LECTURE ON OSTEOTOMY FOR RICKITIC DEFORMITY.

Delivered at St. Thomas's Hospital, January 11, 1894, by

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GENTLEMEN,—I have selected this particular subject for my lecture to-day for several reasons, but chiefly because I think my experience in the wards has shown that when men are asked what they would do in a case of very marked osseous deformity, they immediately reply "osteotomy." Now there are very grave reasons for thinking that this is a mistake unless certain considerations have been duly weighed—namely, those of age, and the presence or absence of general rickets. It comes, therefore, to be a question of the selection of cases before you determine to do osteotomy. Considering, therefore, how important it is to recognize the inadvisability of osteotomy unless it is absolutely required, I have thought that it would be good for me to give a clinical lecture on the subject.

You will understand that I am not now speaking of osteotomy for all cases of deformity, but only for those due to rickets, cases, that is to say, in which there is evidence of either past or present rickets in the bones which has produced a deformity

only to be rectified, so far as you can see, by osteotomy. In some of these cases the operation is immediately required, in others it should be delayed for a certain number of months or years, according to the circumstances of the case. I wish to mention, as far as I can, the points which I deem important.

To begin with, I do not think that osteotomy ought ever to be done under the age of six years. That, I think, is a most important rule to observe. The reason for this rule is that a very large proportion of these rickitic deformities disappear as the child gets older. If I were to perform osteotomy in all the cases that you see come to me, I should be doing osteotomy almost every week in the year. It is quite clear that in many of these cases the deformity disappears, and many come back to me showing a marked improvement. On the other hand, of course, some go on and get worse, and osteotomy has to be performed. Age then is the first and most important consideration to be kept in view. Time must be allowed for the deformity to correct itself by the natural growth of the bone. The deformity is one which, as you know, depends upon two conditions as far as the long bones are concerned—one which produces a general curvature of the shaft of the bone, and the other in which there is an irregular growth in the epiphysis itself. Supposing you have in the shaft of the femur a very marked curve, this alone will produce a deviation at the knee-joint, and yet there may not be much the matter with the epiphysis itself; on the other hand, with the curvature or without it you may have an overgrowth at one side of the epiphysis producing a deformity of the knee-joint. On a diagram I can easily show this. In one of these bones represented in this case of genu valgum, probably there is an overgrowth at one point on the inner side producing a projection downwards of the patient's inner condyle; you may also have the same condition in the tibia, exaggerating the condition still more. On the inner side both of the femur and tibia, then you have an excessive irregular growth, so that the knee projects inwards, and the centre of gravity will no longer pass through the joint. This is the condition known as genu valgum, and may be due either to curvature of the femur itself, or to the irregular growth at the epiphyseal line. The point now is to recognize when you examine a case, at whatever age it be, which of these two conditions exist, or that both exist. Having made up your

mind that it is dependent to a large extent on the curvature of the bone, and not, to a great extent, on irregular growth at the epiphyseal line, your duty is to wait. Many of these curves correct themselves easily in the process of time. In the case of the epiphysis, on the other hand, it does not so readily rectify itself as in the other instance, and is likely to increase. Supposing the child to be under six years of age, you would postpone the operation till after that period. At two years of age, for instance, you would not treat the case by operative procedure, but on general principles. You would send the patient into the fresh air as much as possible; and if poor you should make arrangements by which they can be boarded out in some country village. This is not a difficult matter at the present time. There are Societies in London which undertake to plant out such children amongst families in the country. There the general condition improves, and the deformity may thus disappear. The bone straightens out again, the growth becomes regular, and the limb assumes its normal position.

One other point with regard to the examination of the case has to be considered, namely, whether the child is the victim of general rickets in the bones—not rickets as a general constitutional condition, manifested, for instance, by intestinal trouble, enlarged liver, etc., which would come under the medical description of the case; but whether the child has enlargement of the epiphyseal ends of the bones all over the body. If this be the case, it is still more essential to treat it on the lines I have just mentioned by postponing operative treatment.

We pass now to the details of the operation. We will begin with the knee. We will assume that the age of the child, the length of time the condition has existed, and the ossification that has already taken place in the rickitic parts rendering improvement little likely to occur, point to the necessity for operation. It has been too much the habit to consider division of the shaft of the femur just above the epiphysis as all that was necessary. I am constantly pointing out in the wards cases over ten in which there is a ridge in the centre of the epiphysis itself. It is a very marked condition which everyone ought to recognize, because it is a very good indication of the position for osteotomy. Usually the osteotomy is a simple one above this line or ridge. But there are a few cases in which that is not sufficient; and it depends upon your

keenness in diagnosing which bone is chiefly affected as to whether you should do an osteotomy of the femur or of the tibia. In certain cases there is very little doubt that the tibia is as much at fault as the femur. I have had some cases recently, one of which I shall detail presently, in which I have done an osteotomy of the femur, and then had subsequently to do osteotomy of the tibia. If you bend the knee-joint you can feel the line of articulation and the interval between the two bones, then you can tell whether the tibia is as much or more deformed than the femur. If the former is at fault you will find that the inner head of the tibia runs up obliquely, showing that if you divide the femur alone you will not get the limb into a straight position. Ordinarily speaking, however, the division of the femur above the epiphyseal line is all that is necessary. You make a short incision, either on the inner or outer side, as you like. You are aware that for myself I generally stand on one side of the patient and do the operation on the outer side of one limb and the inner side of the other. It makes no difference which side you select. A short incision in the axis of the limb having been made on to the femur, a sharp chisel is introduced and made to work its way through from one side of the bone to the other with the aid of a mallet. The difficulty and danger of the case consists in letting your chisel go beyond your control; therefore grasp the chisel very firmly in your left hand, and tap with more or less vigour, but always feeling that you have the chisel firmly in hand, so that it will not go beyond the point intended. The anterior cortical bone is easiest to divide; but when you are doing the posterior surface of the shaft of the femur, there is a little anxiety as to whether you may not be near the big blood vessels. But that is a point of which you can only judge by seeing the operation done. Having reached the opposite side, you lever the chisel with your left hand across the axis, not in the length of the limb. You generally know by the curious resonant sound when you have reached the cortical bone at the opposite side. Having divided as much of the bone as you think necessary, you take away the chisel and fracture the bone with your hands. It is most easily fractured by increasing the deformity; whereas, in the opposite direction it is accomplished with difficulty. An antiseptic dressing to the wound, and a plaster of Paris mould to the whole length of the limb will complete the treatment. The other knee may be

treated in exactly the same way, and put up at the same time.

Supposing you are aware that the epiphysis alone is not the cause of the deformity, and that there is also curvature of the femur, ought you to do an osteotomy on the shaft of the femur? Well, I have tried to emphasize the fact that as far as possible this should be left alone; and you will see now a case of mine in the wards in which the deformity of the knee-joint is absolutely and perfectly corrected, but there is, compared to the length of the limb, an enormous curve of the femur. I shall leave that alone. The only way to straighten such a femur is to do several osteotomies all the way down the shaft, and that is undesirable.

You are aware from what I have said that this is the usual operation. Supposing, however, that you have not corrected the deformity, that it is still markedly deformed, and that you cannot get it straightened without bending the femur at the line of section in a way difficult to explain, but easy to see, then you may have to divide the tibia. In this you adopt a different method altogether. In that position it is not desirable to use the chisel; it would be extremely difficult to do so. The skin, of course, is in close contact with the bone; if you had to use the chisel you would be liable to all sorts of trouble and difficulties. Personally, therefore, I have generally used the saw. You make a free open wound right across the front of the leg just immediately below the tibial epiphysis. This incision, about two inches long, having been made across the axis of the limb, the saw is introduced and the bone divided. The fibula is treated in the same way. You have then done three osteotomies in one limb. I shall mention a case in which I did six osteotomies in an afternoon in the same patient. If you make up your mind to do an osteotomy for genu valgum, you must do it so as to correct the deformity. I am loth, indeed, to recommend operation for children; but when you do it do it thoroughly.

A point or two arises with regard to the subsequent treatment. Of course, the case should be perfectly aseptic; there should be no rise of temperature, and no suppuration; and the wound should heal by first intention, requiring, perhaps, only one dressing. Is it desirable, after the operation, when the wound has healed, to make the child wear an iron apparatus from the hip joint to the boot for, perhaps, a year. Well, opinions differ on this point. Till quite recently

I have invariably given irons after the case was at an end from the operator's point of view; but I think your procedure in this respect must vary with each case. Some of them really require irons because afterwards the reverse condition, genu extorsum, is apt to follow. That undoubtedly does sometimes slowly arise after an osteotomy. The case from which the diagram, which you see here, was taken was one in which no operation was done: but I had a diagram made to show this particular variety of genu valgum, namely, genu valgum of one limb, and genu extorsum of the other, a combination of deformities which is occasionally seen. However, the subject we are now discussing is whether you ought to apply an apparatus of iron or steel to the whole length of the limb, from hip to foot, after the operation of osteotomy. If the case be one of a big heavy child I generally order an apparatus; on the other hand, if the child is over six years of age and the parents are sufficiently intelligent, I am inclined to leave it alone for several months, say six months, and see if there is any tendency shown towards deformity. There is this great disadvantage about irons, that they act as a weight and impede healthy natural exercise of childhood. If the patient is, as many of them are, a feeble child, it does not get about so easily as it would without a support of that kind. With regard to such apparatus, therefore, each case must be decided according to the circumstances of the child, the class of life in which it is placed, its general nutrition, and its age.

We come now to another class of case upon which, perhaps, you may not have seen many operations, but in which, I am sure, you have seen many of the deformities. Here is a specimen which shows what I mean, an anterior curve of the lower third of the tibia. It is also beautifully shown in this macerated specimen in which you will see that on this particular curve, as the child gets older, there is a marked bony growth on the side of the concavity which evidently becomes sclerosed. If that be so it apparently contradicts what I have said as regards these bones becoming straight again; because you will assert very truly that if it is allowed that there is bone formation on the concavity which ossifies and becomes sclerosed there is very little chance of its straightening out again. Well, the point is this: I believe that if the patient has arrived at the age of six and the condition has been there

for three or four years, you are right; but if, on the other hand, it is a comparatively recent thing, it may grow straight again. It is the rarest thing in the world to see an adult patient with this particular curve; and yet rickety people do not all die; a very large number of them have had these curves in the lower third of the tibia; it is quite a common thing; and yet a few years ago, twenty years ago, for example, osteotomy was not done for these cases. Still, you rarely see adult patients with this particular deformity.

This curve in the lower third of the tibia is generally forwards and outwards. If it has been in existence for several years you ought to do an osteotomy; on the other hand, if it is not of more than a year's standing, postpone operation if you possibly can. Something also depends on the degree to which the condition has attained. It would be absurd, for instance, to suppose the bone would straighten out in this case. That would be an indication for osteotomy. Still, I would wait till the child was six years old before doing it.

As for the operation: Most surgeons have, I believe, till recently, at any rate, thought it wise to remove a wedge of bone. I am sure I have seen that done most frequently. Macewan, of Glasgow, who is the pioneer, one would say, in osteotomy for rickitic deformities, adopted the wedge, taking out a piece of bone with the base of the triangle to the front, the apex to the back. As time has gone on, however, it has been shown that, with simple section of the bone and then considerable traction on the leg, you can get all the result you require, namely, straightening of the tibia. There is produced behind, of course, a gap, but that is filled up with new tissue, and the limb is straightened without in any way sacrificing the length of the bone. All my recent osteotomies of this part of the tibia have been done by simple section of the bone.

What instrument should be used in this case? Whatever opinions you may hold with regard to the instrument for osteotomy of the tibia above or of the femur, I am quite sure that the chisel is out of place in the lower third of the tibia. I employed it once, and had to use such an amount of force that was really, to my mind, serious. The result, too, was unsatisfactory, as I got necrosis. I have seen the epiphysis at the lower end undergo necrosis over a large area after osteotomy with the chisel. Therefore, I am inclined

to think the operation here ought always to be done with the saw. If you operate at the period of which I am now speaking when sclerosis has already ensued on the concave side of the deformity, you have to use a great deal of force. It is not in the least like an osteotomy for an epiphyseal deformity. You will remember that in the curvature of the femur I did not recommend the operation at all in the shape of an osteotomy. As a rule they straighten again; and if it should not, the deformity is not very obvious. Therefore they are generally best left alone. But in the lower third of the tibia, if you determine to perform osteotomy, you must remember that by the time you proceed to the operation there is this extraordinary development of dense bone on the concave side, so that it is more or less straight behind, but with a tremendous curve in front, the straightness being produced by the development of new bone and its subsequent condensation. This sclerosed bone cannot be divided by the chisel without very great danger. Therefore use the saw. Make a free wound in front. It is of no use attempting to do what is called a subcutaneous operation. "Subcutaneous surgery" is really no longer the term we use for these operations. We do not in the least fear an open wound with the precautions now always adopted. We make an incision right across the tibia on its anterior aspect, introduce the saw, and divide the bone. Even with the saw you will sometimes find it difficult to divide the bone cleanly, it takes a good deal of time and force to get through the sclerosed bone. One special kind of saw is particularly serviceable for this purpose; and if you determine to take away a wedge it is almost essential that this instrument should be used, namely, Gowan's saw, which is connected with a pair of forceps. The shaft of bone is held by the forceps whilst the saw is in action. This particular instrument should be used if you take a wedge from the bone; but if you adopt the simple section I am recommending, it is not necessary. The case is afterwards treated in the same way as other osteotomies; the wound is sutured and dressed, and plaster of Paris applied to the whole length of the limb. As a rule there is no necessity for irons or any support afterwards. When really firmly united, as far as my experience has gone, there is very little chance of subsequent deformity. On the other hand, one must bear in mind that there are cases recorded

—I have seen one—of ununited fracture after this osteotomy, which are believed to have arisen from the sclerosed condition of the two divided ends. The sclerosed bone being less vascular, less easily throws out plastic material for subsequent ossification. You will, therefore, readily understand that you must get perfectly firm union before you dismiss the case; and when it is firmly united you may, as a rule, trust to nature to do all that is necessary.

Let me now rapidly allude to a few cases that have been under my own care during this last year.

The first case occurred in the early part of 1893, being admitted on February 23rd. It was that of a lad *æt.* 19. This age (and I will speak of another nearly as old) is long beyond the period at which any question as to the necessity of the operation would arise. Although he was 19 the lad looked scarcely older than 12, being almost dwarf-like. He had well marked and still active rickets everywhere in his bones; every epiphysis was enlarged with well-marked ridges on them, showing that the rickitic changes had not ceased. The patient had been under my care two or three years before for genu valgum. I had divided his femora and sent him out with perfectly straight limbs, and with an iron support which he had worn till within six or nine months of coming again. He had this time just the same genu valgum, with six inches separation between the malleoli. In my former notes I find he had seven inches between the malleoli, while on dismissal from the hospital they were close together. I could not find either from himself or his parents, whether or not the separation had arisen after he had discarded the support given him. On examining the case thoroughly it was obvious to anyone with experience of these cases, that the femur was no longer responsible for the deformity. If the routine operation of osteotomy for genu valgum had been done the femur would have been divided, and this would really have done considerable damage. The deformity this time was due entirely to an irregular growth at the tibial epiphyses. I therefore divided the tibia immediately below the epiphyseal line, and the deformity was at once corrected. The boy went out with straight limbs, and is wearing iron supports. The deformity is now practically corrected. I mention this case because it is a good illustration of the occasional necessity of dividing the tibia as well as the femur in rickitic deformities.

The next patient was a child 5 years and 9 months old. You will at once say that in operating on this case I departed from the rule I laid down that the child should be at least six years old before osteotomy is done. Well, this child was within three months of that age, and one may be allowed that amount of latitude. Moreover, the child had had this deformity for nearly four years; it had been noticed since the child was two years of age; and having existed for that time I thought it justifiable to do the operation. The child is in the Victoria Ward now, and is an example of the curious results that follow. The child's bones were markedly rickety all over. There was extreme curvature of the femora, but also irregular growth at the epiphyseal lines; therefore I did the operation at the knee, dividing the femur by the chisel in the ordinary way just above the epiphysis. You will now see that both the limbs from the knee down to the ankle are parallel and in contact. Before, there were six inches of separation between the two malleoli; that has been absolutely corrected. But if you look at the child as it lies on its back in bed you will see that it has the same femoral curves as before; and it does not look at all pleasant. It seems a tremendous bow in both thigh bones. That I shall leave alone. The only thing that could be done would be a multiple osteotomy of the shaft of the femur; but I am sure this condition will improve, if not absolutely disappear in the course of a few years. If not, one can still do what is necessary: I do not think, however, it will ever be necessary.

The third case is also still in hospital,—in Edward Ward, that of a boy, *æt.* 14. This youth had had deformity as long as he could remember. He belongs to the class of case which is spoken of as "adolescent rickets," just as the first case did. Adolescent rickets may be a new development, that is, it may arise at puberty, or on the other hand, it may be a prolongation of the infantile condition. It is shown chiefly by the very large bone ends and the ridges on the epiphyseal lines. This boy had these conditions. He had extreme deformity, the separation of the ankles being over six inches. He was constantly tumbling, and had great difficulty in running and walking. It was quite clear that at his age the deformity was unlikely to be corrected by any improved growth at the epiphyseal line, and therefore the operation was performed. Flexion of the knee-joints showed

that there was tibial as well as femoral deformity. I therefore did six osteotomies, dividing both femora, both tibiae, and both fibulae. The result has been perfectly satisfactory. He has straight limbs from knees to ankles. I shall probably consider it better on the whole, to send him out with irons. He still shows the extraordinary curves in his femora, which I do not think it is desirable to correct.

The last case is that of a child, aged 6, admitted in July last, with deformity in the lower third of her tibiae. There was no very marked rickets about the bone ends, and the child was healthy. The condition had existed for four years. It was therefore unlikely to be corrected for a considerable length of time, and, inasmuch as the operation is fairly safe, I did a simple section of the tibia. If it had been necessary to remove a wedge, I doubt whether it would have been wise. A simple osteotomy across the lower third of the tibia is hardly a dangerous procedure. I corrected the deformity very easily. Considerable traction was necessary to draw the limbs into a straight line. When that was done the deformity was corrected. The only difficulty consists in maintaining enough traction with the plaster of Paris and the voluminous dressing that is applied. When the wound is healed you may reduce the dressing to something insignificant, and the plaster of Paris then fits so accurately to the foot and knee, that you can employ as much traction as you like, and so prevent the heel from being drawn upwards or the foot backwards by the strong calf muscles. I saw this case the other day, and the legs were quite straight.

Osteotomies, then, in my opinion, should not be done in a wholesale way. In the wards I have noticed that if I show such a deformity to men going up for examination, and ask, What would you do? they immediately reply, Osteotomy. It is for that reason I thought it desirable to say a few words about the operation.

Application for Burns:—

R Tr. Opii ... 3iij
Aqua Laurocerasi ... Oj
Glycerini ... Oij

M. Ft. applic. Sig.: The burnt parts to be kept constantly moist with the application.

(*L'Union Med.*)

CLINICAL NOTES.

(Specially reported for *The Clinical Journal*. Revised by the Author.)

WITH DR. LEWERS AT THE LONDON HOSPITAL, JANUARY 10, 1894.

A Case of Contracted Pelvis.

This patient, Gentlemen, is a woman, who was under my care in Davis Ward some years ago, on account of having an unusually large cystocele; her case is, however, more particularly interesting because she has a contracted pelvis. The external measurements of the pelvis are as follows:—

Between the anterior superior iliac spines = $10\frac{1}{2}$ in.
Maximum distance between the iliac crests = $10\frac{1}{2}$ in.
External conjugate (*i.e.*, from the last lumbar spine to the front of the symphysis = $6\frac{1}{2}$ in.

Internally, measuring from the sacral promontory to the under-border of the symphysis pubis, by means of two fingers of the right hand introduced into the vagina, we find this distance to be $3\frac{1}{2}$ inches. This measurement is called the diagonal conjugate. For practical purposes it may be said that if half an inch is deducted from the diagonal conjugate, we obtain the value of the true conjugate. In this case we thus find that the conjugate of the brim measures about $3\frac{1}{2}$ inches.

If we get the patient to stand up you will see that she is considerably below the average height, and this alone should at once lead you to suspect the possibility of some pelvic contraction, and to make the measurements necessary to ascertain whether this is so or not, if you see the woman when there is a prospect of her becoming pregnant. In the case before us the patient is now past the menopause, but the history of her confinements is instructive. She had five confinements, on each occasion going to full term, and on each occasion she tells us the child had to be destroyed in order to effect delivery.

Incidentally, I may tell you that the measurements indicate that here we have to do with a flattened rickety pelvis. It is a "simple flat" pelvis; that is to say, the contraction is limited to the conjugate of the brim, and there is no reason to suppose that there is any contraction in the transverse diameters of the pelvis. You may know that the flattening is due to rickets from the

relation of the measurement between the anterior superior spines to that between the crests. You notice that in this case the former is nearly the same as the latter, instead of being, as normally it should be, about an inch less. The alteration in the relation of these measurements points to rickets. Apart from this there are also indications of rickets in the patient's legs.

Now let us consider what methods of treatment may be adopted in a patient who is pregnant, and has the degree of pelvic contraction present in this case. In the case of a woman pregnant for the first time, you may choose to let her go to full term, trusting to be able to deliver her of a living child by forceps or turning. Quite recently I was called in to a case of labour at full term in a primipara with a pelvis having somewhat similar measurements; but you will see that the transverse external measurements are less than normal, and indicate some lateral contraction as well as flattening; in fact, it was a flattened pelvis with slight general contraction. The measurements in the case I refer to were as follows:—

Between anterior superior spines ...	= $9\frac{1}{2}$ in.
Maximum distance between iliac crests = $9\frac{3}{4}$ —10 in.	
External conjugate	= $6\frac{1}{2}$ in.
Diagonal conjugate	= about $3\frac{1}{2}$ in.
Conjugate of brim therefore...	= about 3 in.

In that case I succeeded in delivering her of a living child by means of the forceps, although with a great deal of difficulty; both mother and child did quite well. There are, however, two things to be said about this case. One is that it was a first pregnancy, and the size of the child increases in successive pregnancies. The other is that the child was a girl, and therefore probably smaller than if it had been a boy. Therefore in this case, for both these reasons, it is quite likely that in a subsequent labour it may be impossible to deliver her of a live child in the same way as on the present occasion.

To return to the case before us. With a pelvis such as she has, there would be several methods to choose from. You might, for instance, induce labour at about the thirty-second week with a fair prospect of delivering a live child; the objection to this plan is that, although many premature children are *born alive*, a very large proportion of them do not long survive their birth. If, on the other hand, you decide to let such a case go to full term, you may possibly deliver her of a living

child by the forceps, or perhaps by turning; although where the survival of the child is specially in view, turning is a much less hopeful procedure than delivery by the forceps. For my part, in such a case, if I failed to get the head to engage after a thorough trial with the forceps, I should perform symphysiotomy—an operation introduced towards the end of the last century, which, after being abandoned for many years as useless and highly dangerous, has been recently resuscitated, and with very good results to both mother and child. Some of you have seen the patient on whom I performed symphysiotomy last February in this hospital. In that case the indication for the operation was more especially that I was unable to deliver with the forceps, although it was very thoroughly tried, and also the fact that the child was alive. There was also moderate pelvic contraction. After dividing the symphysis, I found it quite easy to effect delivery with the forceps, using only one hand, and sitting down before the patient. I may add that both mother and child did well. It may interest you to know that this was the first successful symphysiotomy in England, and the first operation of the kind performed in England for more than 100 years.

A Case of Metrorrhagia.

This patient has been sent up with a note from her medical attendant asking me to investigate the case. She is 51 years old, and has had one child 18 years ago, and no miscarriages. She complains of having had pain at the lower part of the back and in the front of the abdomen, also in the left iliac region, for three years. The chief cause of her coming, however, is on account of bleeding. She has had a blood-stained discharge from the vagina almost continuously for eighteen months. The discharge is said to have been offensive for the past week. There is no very distinct history of the menopause having occurred, but she tells me that about two years ago there was a period of two or three months during which she "saw nothing." The significance of the metrorrhagia would have been increased if there had been a distinct history of the menopause having occurred before it began. For, as I frequently have occasion to say in this room, in a very large proportion of cases, hæmorrhage commencing after the menopause is due to cancer—it may be of the cervix, or perhaps of the body of

the uterus, or it may have begun in the vagina, or on the external genitals.

On proceeding to examine this patient we find nothing abnormal in the abdomen, but now that she is lying on her left side you see there is a good deal of recent blood about the external parts. On vaginal examination I find two little polypi each about the size of a small raisin attached by a pedicle about half-an-inch long to the posterior lip of the cervix; these, however, are quite inadequate to account for the amount of blood that is coming away while I am examining her. The cervix is patulous, and I can pass my finger easily through the internal os. I then feel a smooth, firm, and somewhat fleshy body in the cavity of the uterus. I think it is probably a mucous polypus growing in the body of the uterus; you know that such polypi are much commoner in the cervix, still they do occur in the endometrium, and I think we have an example before us.

There are, at least, two other conditions that should be considered when we have a case with hæmorrhage and a patulous cervix with a firm body to be felt at the internal os just within reach of the finger. The first I refer to is abortion. Many cases with such physical signs are cases of abortion in process of becoming complete, or of molar pregnancy where the mole is about to be expelled, or of incomplete abortion, where some part of the ovum has been left behind. In this case, however, the persistence of the hæmorrhage for eighteen months, as well as the fact that she has not been pregnant for eighteen years, and the fact that she is 51 years old, are points strongly against the supposition that her present trouble is connected in any way with pregnancy. Nor does the substance at the internal os feel to me like anything that has probably originated in a pregnancy.

The other condition I refer to as one from which the present case must be diagnosed, is cancer of the body of the uterus. The patient's age and her relative sterility would be quite in keeping with her present trouble being cancer of the body of the uterus. Here again the substance felt at the internal os is not quite the kind of thing one feels in cancer of the body with a fungating growth occupying the endometrium and presenting at the internal os. This growth is firm and movable, suggesting that it has a narrow attachment. Cancerous growths in the uterine body, when they are to be felt at the internal os, may, or

may not, have a somewhat polypoid form, but they are characteristically soft and friable.

In this case I should advise the patient to come into the hospital soon. She should have an anæsthetic, and then one could more thoroughly examine the relation of the fleshy substance felt presenting at the os uteri. The cervix could be drawn down with a volsella, and I have no doubt the finger could then be passed up to the fundus. If it is only a mucous polypus it could then be easily seized with strong polypus forceps and twisted off, the uterus being washed out with an antiseptic solution; and, no doubt, nothing more would be needed. If, however, the growth seemed of a suspicious nature, and further examination of it showed it to be cancerous, the question of removal of the whole uterus would have to be considered. My own expectation, however, is that the growth will prove to be non-malignant.

[This opinion was confirmed at the examination under chloroform about a week later. The growth proved to be a mucous polypus, growing in the body of the uterus, near the fundus.]

A Case of Ovarian Tumour.

This patient has been sent up to me for an opinion by her medical attendant. She is 22 years old, and has had one confinement four years ago. Instruments were used, "and she was sewn up afterwards." She says that she has not been well since the confinement; she had "ulceration of the womb" for twelve months. Her present trouble dates from eight weeks ago, when she noticed a good deal of pain in the lower abdomen, and rather at the right side. She did not know of there being any swelling in the abdomen till her doctor told her of it some ten days ago. Her chief complaint has been on account of the pain. She was quite regular till two months ago, but has not "seen anything" since then. On examining the abdomen you can see that there is considerable prominence over its lower two-thirds, and when she takes a deep breath you observe, on expiration, a distinct suggestion of an outline, as if the abdominal wall were adapting itself to an underlying tumour. On palpation one feels a tumour, evidently containing fluid, rising from the pelvis and extending four fingers' breadth above the umbilicus; the upper part of the tumour feels harder than the

rest, and probably corresponds to a solid portion of the tumour. Nothing is to be heard over the tumour. After the patient was turned on her left side a catheter was passed, but only some two ounces of urine were drawn off. The external parts are not specially blue. On vaginal examination one feels the vaginal portion of the cervix to be no larger than normal, and not specially soft. In front of the cervix a hard body can be felt like the body of the uterus when of the normal size, and on tilting the cervix this hard body moves with it. There is no doubt, in fact, that it is the uterus not enlarged. To the left, and separated from the hard body by a narrow interval, is a bulging elastic swelling continuous with that felt in the abdomen. Through the speculum you see the cervix is not blue. On attempting to pass the sound I find it only goes as far as the internal os, and that further pressure merely pushes the whole uterus upwards. We must, therefore, expose the cervix with Sims's speculum and fix the cervix with a tenaculum. When this has been done you see the sound now passes without much difficulty with the curve forwards a distance of two and a half inches. We have now enough material for coming to an opinion, and I have no doubt that the case is one of ovarian tumour.

A Case of Apparent Occlusion of the Vagina and Urethra following Labour.

This is a very curious case. She is 24, and the history is that she had a child four months ago. It was an eight months' child. The labour is said to have been easy. Since the confinement she has menstruated regularly. Her complaint is that since the confinement her husband cannot have connection with her.

There is nothing abnormal to be felt in the abdomen; but, on examining the patient locally, you see that there seems to be no opening either to the vagina or the urethra. There being, apparently, a uniform fleshy surface unbroken by any opening, extending from the vestibule to the skin of the perineum. As, however, she tells us she menstruates regularly, and passes her water properly, though with difficulty, it is certain there must be communication between the vagina and the outside, as well as between the urethra and the outside. You see, however, that even by a careful examination I cannot, for the moment, detect the openings in question. The right thing will be for

her to come into the hospital and be thoroughly examined under chloroform and in a good light. We shall then probably find some minute openings. The appearance of the vulva is very similar to what one sees in cases of congenital occlusion of the vagina, except that in such cases the opening of the urethra is normally situated. It so happens that a case of congenital occlusion of the vagina is under my care in the hospital at the present time.

Mitral Stenosis.—Dr. James Barr advocates the use of Atropine and Nitro-Glycerine in combination, in this form of valvular heart disease.

R. Liq. Atropinæ Sulph. ... ℥vj
 Liq. Trinitrini ... ℥vj
 Aq. ... ad ʒiss

M. Ft. Mist. Sig.: ʒj to be taken three or four times a day.

(*Liverp. Med.-Chir. Journal.*)

REVIEWS.

Year Book of Treatment. (CASSELL & Co., 1894).

To busy practitioners who require all the latest information on the ever-growing branches of our profession, this volume, the tenth of its series, will be very welcome. The book consists of brief epitomes of the best of the recent suggestions in therapeutics collected from numerous sources. The compilers are in great proportion the same as last year, and they have done their work again with excellent judgment and care. A new feature in this year's volume is the re-introduction of an article on diseases of children, which has been admirably drawn up by Dr. Dawson Williams; the section on Bacteriology as a helpmate to therapeutics will, we hope, show us more available results in time.

We have received from Messrs. Kelly & Co., of 184, High Holborn, their *London Medical Directory* for 1894. This is the sixth annual appearance of a similar volume, and its contents are now so well known as to require little criticism from us. The present number is fully up to its predecessors in accuracy of information and readiness of reference to any name, but the peculiar arrangement of the street list is unfamiliar and a little puzzling at first. The list of nursing institutions in London is a most valuable addition to the *Directory* for those requiring a nurse in a hurry, and, as we believe, a feature unique amongst Directories.

THE CLINICAL JOURNAL.

WEDNESDAY, FEBRUARY 21, 1894.

THE CARE AND TREATMENT OF DIABETIC PATIENTS.

By ROBERT SAUNDBY, M.D., F.R.O.P.,

Professor of Medicine, Mason College, Birmingham ;
Physician to, and Lecturer on Clinical Medicine in, the
General Hospital, etc.

GENTLEMEN,—Permit me to say a few words by way of introduction, upon the objects of these weekly conferences, and the place they should hold in the general scheme of medical education. Premising that you are well grounded in anatomy, physiology and pathology, and that you possess some general acquaintance with the principles and methods of natural science, especially chemistry, you attend the practice of this hospital with the purpose of learning by actual daily observation and experience the course of diseases and the phenomena presented by the subjects of them during and after death ; of seeing for yourselves therapeutic principles practically applied, and of estimating their value ; in a word, of laying the foundation of your clinical experience, upon which you will continue to build during the remainder of your professional lives. It, therefore, does not need argument to prove that this is a very important part of your career, and that a great responsibility rests upon your teachers as well as upon you to see that this precious time is devoted wholly to the proper purpose of your coming here, and that it is not wasted by futile repetitions of mere theoretical teaching, such as you have already received, or will receive, in the systematic courses of lectures delivered at the college, or in aimless wandering about the wards like a tourist in a picture gallery, understanding little that you see, and seeing far more than you can possibly store up in your memories. I believe that each student should, throughout his hospital course, be constantly attached, for a longer or shorter time, to some special department. You are compelled, by the regulations of the General Medical Council, to be ward clerks and dressers, but you should also find time to assist the pathologist, to take a share of the work in the special departments for diseases of women,

throat and ear ; to attend at the eye hospital, etc. If you will go regularly you will find that you will be very welcome, that you will soon become of use, and that you will learn a great deal. It is the casual student who learns nothing, and who only hinders the work of a busy out-patient department. I am certain, if you will bear this advice in mind, and each three months pursue a definite object, you will learn much more than by desultory attempts to follow the whole practice of the hospital during the same session.

The part played by these lectures is not so important here as in larger schools, where it is impossible for a dense crowd of students to be taught in the wards around a bedside, but our intention is to give you a more complete demonstration of a case, or a series of cases, than can be done during a ward visit, and to add to your clinical knowledge and experience by drawing your attention to examples illustrating points of importance in the clinical history, pathology, and treatment of disease.

The patient I have to introduce to you to-day is a man, W. H., æt. 29, who was admitted into hospital on December 27th, 1893, complaining of being "all the while hungry and thirsty" and of loss of flesh and strength. He is, as you see, a spare man, weighing 10 st., showing a considerable loss of weight, as he measures 5 ft. 7½ in., and as lately as September last scaled 11 st. 9 lb. He tells us that his illness, which first attracted his attention by the increase in the quantity of his urine, began a year ago, but has been much worse during the last three months, his strength becoming so much diminished that he became unable to continue his work as a builder's labourer. He is an unmarried man, who has lived a somewhat intemperate life, but he denies having had any previous illness, including syphilis, or having ever met with any serious personal injury. He has not suffered from rheumatism or gout, nor does he present any signs of these diseases. His family history presents, positively, the death of a sister from consumption and the early death of his mother from some unknown cause ; but these facts, though suggesting the presence of a predisposition to tuberculosis, which undoubtedly occupies a frequent though

obscure relation to diabetes, are deprived of some of their significance by the sister's husband having died of phthisis three years before his wife. It is possible that she may have been infected by him, as there are indubitable cases on record where a healthy wife, coming of a family without known predisposition to tuberculosis, has become infected by a consumptive husband.

Negatively we find that he knows of no case of diabetes in any relative. On examination his teeth are good, his tongue is large, furred, and flabby, while his mouth is dry, he complains of a sweet taste in it, and on admission he was very thirsty. There is no distinctive odour in his breath; his bowels are regular, and the physical signs and appearance of the abdomen are normal. His skin is soft and moist, and he complains of sweating profusely at times, chiefly in the night; but he has no cough, and the only change to be detected in the lungs is some interrupted (jerky) respiration at the right apex. The heart's apex is in the fourth left intercostal space, half an inch internal to the vertical nipple line, but its area of dulness is not increased, and the sounds, though feeble, are unaccompanied by any murmurs. His pulse is 60, feeble and easily compressible, its characters being illustrated by the pulse-tracing exhibited, which shows a short, feeble upstroke, with a gradual falling away from the summit and ill-marked dirotic notch.

I am in the habit of attributing considerable prognostic significance to the state of the pulse in diabetes. The excess of sugar in the blood is the occasion of sufficient obstruction to the circulation to cause an increase of the intra-arterial tension so long as the heart acts with normal vigour; but as the disease progresses nutritional changes affect the muscular structure of the heart, and with their development the pulse becomes feeble and easily compressible, thereby becoming an index of the wasting heart, and a prognostic sign of the most unfavourable kind.

On his admission our patient was passing 126 ounces of urine, containing 5 per cent. of sugar, but he was, and had been for some time, under medical treatment and dietary restrictions.

The urine gave no reaction with Ferric Chloride, and contained only a faint trace of acetone when tested with Nitro-prusside of Sodium and Ammonia. His eyesight is good, and ophthalmoscopic examination reveals nothing abnormal; his knee-jerks are absent, but in other respects he presents

no evidences of any disease or derangement of function in his nervous system.

This is therefore a moderately acute case of diabetes in a young man, without family predisposition, in whom there has resulted great loss of flesh and muscular vigour, including especially the heart muscle. It is not a favourable case for two reasons; the first is a general one, and applies to all cases of diabetes which occur before middle life; we may say that the more a patient is under 40 years of age the worse for his prospect of life, and the more he is over 40 the better his chances under suitable treatment to prolong his life, and possibly to throw off the disease altogether. The second reason is a special one, and is founded upon my opinion of the state of his heart.

What is the change in the heart muscle to which I attach so much importance? It is fatty degeneration of the muscular fibres, which waste and are replaced by simple fibrous tissue. The loss of knee-jerks is also a sign which has been empirically established to be of bad omen.

It is not a case in which we can hope to do a great deal; we must not expect to cure him; but it is our duty to place him under the most favourable conditions within our power for preserving his life and controlling the disease. This leads me up to a subject I desire particularly to talk to you about, namely, the care and treatment of individual diabetic patients. It is too much the custom to think and to act as if all that can be done for a diabetic patient is to cut off all his saccharine and starchy food, to order him some gluten bread, and to prescribe Codeia, or Opium, or Salicylate of Sodium, *secundum artem*! Nothing can be worse for the patient, no practice can be more irrational and absurd. The phrase *secundum artem* is not necessarily one of bad meaning, but it is so associated with the blind following of devitalized tradition and the justification of the most unintelligent routine, that the art of medicine has declined greatly in our respect. With the growth of our nosology, and the subdivision of disease groups into distinct clinical types, accurate clinical study and the data thence derived for precise diagnosis should afford a sound basis for definite rules, to be followed by the practitioner in the treatment of any given case of disease; but at present this is only possible in certain instances; while in diabetes, the pathology of which is still so obscure, we cannot expect the rule of thumb, by which some would bind us, to be successful.

The first duty of the practitioner who undertakes the care of a diabetic patient is to make a careful examination of him, to weigh him, to have his urine collected for twenty-four hours, to measure the quantity, and to estimate the percentage of sugar. All this may be done without making any alteration in the existing treatment or diet, so long as there are no very urgent symptoms and too much time is not lost. The next point to determine is the influence on the amounts of urine and sugar of a diet from which all starch and sugar are rigorously excluded; that is to say, a diet consisting of meat, eggs, fish, jelly, almond biscuits and cakes, tea or coffee, and mineral water. Such a diet is very difficult to put up with for any length of time, but can be tolerated for a few days. I have told you what the condition of this patient's urine was on admission, viz., 126 oz. of urine, sp. gr. 1049, sugar 5 per cent. On strict diet the amount fell to 50 oz., sp. gr. 1026, containing 1.2 per cent. of sugar. This result showed that the glycosuria was for the most part dependent upon the carbo-hydrates derived from the food, and could be regulated accordingly. It is most undesirable to keep diabetics who are losing flesh and strength on too strict a diet; I may go further and say that *every diabetic should be allowed as much carbo-hydrates as he is able to assimilate, estimating this by the amount of urine and sugar and the body weight.* My observations have proved that milk, not exceeding one pint a day, and potatoes, to the amount of two or three large ones daily, may often be added to the diet with very little if any increase in the polyuria and glycosuria, and with benefit to the patient; I also allow green vegetables, sea-kale, celery, salad, cream, cheese, cream cheese, isinglass blanc mange, and Irish or Iceland moss. I do not recommend gluten bread, as at its best it is nasty and dear, and the most favourable specimens contain 25 per cent. of starch; in fact, with less starch it is impossible to make a bread which can be baked and eaten. Therefore, where a strict diet is to be ordered, I recommend Clark's starchless biscuits, made from almond flour, and in other cases I give a strictly limited amount of bread toasted. It may be asked why toast is better than bread. It is of course not better in the sense of containing less carbo-hydrates, but it is more satisfying and easier to define by weight, so that we may permit in suitable cases two ounces of toast with each meal; but this must be done with

caution, and its effect watched anxiously. Irish moss is an addition to the dietary of diabetics which is of great utility. It supplies a basis for puddings of which a great deal may be made by a good cook. Iceland moss does not make such a nice dish to look at, but is not bad to eat.

In addition to all the foregoing, it is now possible to give diabetics carbo-hydrates in the form of lævulose or left-handed sugar, which can be obtained in two forms. The first is a granular white powder prepared by Schering and Glätz, of Berlin, and the second is a treacle-like substance, produced at the suggestion of Professor J. B. Haycraft, and sold by Messrs. Allen & Hanbury. The second has the advantage of being much cheaper than the other. I have given both forms to a number of patients, the maximum allowance being 1½ oz. daily, in three doses of half an ounce, and as a rule I have not found any noteworthy increase in the sugar excreted. For some reason which I have not been able to determine, the patient whose case we are considering has relapsed, and in spite of all precautions is now passing as much sugar as on admission. These accidents are only to be accounted for in one way, namely, that the patient is getting sugar or starch in some undiscovered manner; and although in the present instance I do not accuse the man of deceiving us knowingly, I am sure that that is the explanation. Therefore, his case is not a very satisfactory illustration of the use of lævulose. He began with six drachms daily on Jan. 1st, when his urine was under 50 oz., and the daily excretion of sugar only about 250 grs. No increase took place on this dose, but on the 5th when the lævulose was doubled he passed 920 grs. of sugar, and on the 9th and 10th over 1100 grs. The lævulose was then stopped, but the sugar next day was over 1500 grs., and rose in the course of the next few days to over 2000 grs. This does not indicate a distinct relation between the lævulose and the sugar increase.

Let me refer to another case. E. H., æt. 14, was a case of moderately acute diabetes of three months duration. He had lost weight; his urine was over six pints and contained 6 per cent. of sugar. On strict diet the urine fell to four pints and the sugar to less than 2 per cent. He was allowed green vegetables, and three large potatoes daily without any ill effects. After taking this diet for eleven days he had gained four lbs. in weight, his urine was from 50 to 60 oz. daily, and the sugar under 2 p. c. He was then given six drachms of

lævulose daily, and three days later this was increased to $1\frac{1}{2}$ oz. He took this for 18 days without change, gaining two lbs. in the time. It was then stopped for a fortnight during which he gained three lbs., and resumed again for the remainder of his stay in hospital, which amounted to three weeks. On his discharge he had gained 12 lbs. in weight, his urine was between 50 and 60 oz. daily, containing 2.5 per cent. of sugar, and he looked fat and well.

These figures show that lævulose may be given without doing harm, but it cannot be given in large enough quantities to constitute an important article of diet. To many patients it is most grateful, as they have a strong craving for sugar in some form, but it is especially valuable because experience shows that the addition of some amount of carbo-hydrates to the diet is a necessity for the patient's welfare, and I have already said it is our duty to give these substances in such forms and quantities as may be assimilable. A recent writer has shown that the addition of carbo-hydrates to the diet will cause in most cases the disappearance of acetone from the urine, so that they act as prophylactics of that dangerous state of the blood in which coma may supervene.

But in order that you may treat your diabetic patients liberally you must watch them closely until you know what they can assimilate. At least once a week you should note their body weight, total daily amount of urine, and amount of sugar, while it is well to see that they are not excreting acetone or aceto-acetic acid, although if you follow the methods I have recommended you will not have to blame yourself should they be present. Let the period of absolutely strict diet be as short as possible, only sufficient to enable you to judge of its effect, and for this three complete days are enough.

The question of what diabetics may drink requires an answer. Tea and coffee, of course, sweetened with saccharine or lævulose, and with added cream or milk,—wherever possible cream should be taken, as it is a good food. Cocoa should only be allowed if free from starch, but diabetic cocoa and diabetic chocolate are supplied in the shops, though I have no experience of their use, English patients preferring tea or coffee.

Malt liquors are generally inadmissible, as they are highly acid, or contain sugar and dextrines, and are liable to cause gout, to which many elderly diabetics are predisposed. But in mild cases I have known bitter ale taken in moderate quantities without any ill effect, so that when desired it may

be allowed experimentally in defined quantity, and the result in the urine watched with care. Light dry wines—of which Bordeaux claret is the best—may be recommended generally to diabetics; they should be taken with alkaline mineral water.

A moderate allowance of whisky or unsweetened gin, not to exceed four ounces daily, well diluted, suits some people better, and is only harmful if there is any gastritis.

A diabetic is above all men an invalid, who carries his safety very much in his own hands. Unfortunately, they too often become tired of taking care of themselves, and pay the penalty of some slight indulgence with their lives. Not for him are festive gatherings and late hours. Neither pleasure nor business should be allowed to sap the feeble structure of his vitality; and above all he should avoid excitement, hurry, and fatigue. On the other hand, he should take plenty of moderate daily exercise in fresh air; his clothing should be warm; his habits regular, and his allowance of sleep liberal. If he can leave home conveniently, he may derive benefit from a winter residence in a more favourable climate than is usually to be met with in England; but the south coast of even this country may be on the whole the best for him, or he may go to the Riviera, or to the Canaries, or to Egypt. In summer there are some health resorts where the doctors make a speciality of the treatment of diabetes, and the large experience they gain enables them to render valuable assistance. These places are Karlsbad, Neuenahr, Vichy, and Contrexeville. There is no special virtue in their waters, but the places are health-giving, and the system pursued, and the medical advisers to be found at the first three deserve our confidence. It is, however, necessary to warn you against sending advanced cases away from home. They stand travelling very badly, the fatigue, excitement and small worries incidental to it operate most adversely, so that too often the patient arrives at his destination to find not health but a grave. Chronic cases which maintain a certain equilibrium, may be sent away with a reasonable expectation of benefit, but those that are doing badly at home under judicious treatment, will do no better away, and by going expose themselves to great dangers.

The drug treatment of diabetes should be rational and not routine; our aim must be to remove certain unfavourable symptoms, not to cure the disease. In trying to do the latter you may injure your patient, while, in caring for your

patient, you will now and then succeed in curing the disease.

Opium is a great aid to proper diet in diminishing the amount of urine. Most patients, when they come first under treatment, complain of broken rest at night from frequent rising to make water. A grain of Extract of Opium at bed-time will, as a rule, cause the disappearance of this trouble, and marked improvement in the patient's general health, appearance and subjective sensations follow as a consequence of undisturbed sleep.

The bowels, in diabetes, are generally constipated, and this reacts prejudicially on the patient by the absorption into the blood of poisonous substances from the intestine, causing lassitude, drowsiness, depression, irritability, and fitfulness of temper. Saline aperients are the best means of overcoming this, either in the simple forms of the Sulphates of Magnesia or Soda, or in the more complex form of mineral waters, among which I prefer Rubinat water, a wineglassful of which, taken fasting in the morning, is a most efficient aperient. If, as commonly happens, the urine is very acid, depositing a large amount of cayenne pepper-like crystals of uric acid, we may counteract the tendency to lowered alkalinity of the blood by giving Vichy or Contrexeville water, which may be taken with wine or spirit at meals, or by the use of a single dose, thirty or forty grains, of Citrate of Potash at bedtime.

These are the most commonly useful remedies, but even they should only be employed when there are indications for their use.

I cannot attempt to discuss here the various complications of diabetes and the treatment applicable to them, but I may say that, in so far as they depend upon the primary disease, they are controlled by whatever means influence it beneficially; they depend greatly upon the patient's general health and hygienic surroundings, and are for the most part to be treated by the application of the general principles laid down in this lecture. The great hindrances to the more successful management of diabetes are routine methods and the neglect to study the individual capabilities and requirements of each case, added to the carelessness, ignorance, and self-indulgence of some patients. The last we cannot help, but within the limits where success depends upon ourselves we have plenty of opportunity to exhibit greater care, closer observation and a nicer adjustment of rational means to well-considered ends.

TWO LECTURES

ON A CASE OF

UNILATERAL BRONCHIECTASIS, With Remarks on the Pathology, Diagnosis, and Treatment of Bronchial Dilatation.

Delivered at St. George's Hospital by

WILLIAM EWART, M.D. Cantab., F.R.C.P.,
Physician to the Hospital.

I.

GENTLEMEN,—The affection which I shall endeavour to describe to you, presents points of analogy with Corrigan's cirrhosis, which formed the subject of a previous lecture;* and we shall have an opportunity of contrasting the two diseases. The case is one of unilateral bronchiectasis, with unusual enlargement of the sound lung and great encroachment of the right pleural cavity into the opposite side of the thorax.

It may add to the interest you may take in this study, if you are made aware that bronchiectasis is a disease of modern discovery, and that it was first observed by a medical student. Laennec† relates that, in 1808, Professor Cayol, then a student, having noticed in two cases a remarkable dilatation of the bronchial tubes, and finding in books no description of this condition, brought to him the two specimens for examination. This circumstance accounts for Laennec having been credited with the discovery, as well as with the first clinical and pathological description of bronchiectasis.

The disease not being fatal in the majority of cases, post-mortem returns are no safe guides to its frequency. Biermer found a percentage of 2, and Willigk a percentage of 4.4 in their respective series of autopsies.‡ Again, its clinical recognition has doubtless gradually improved since the beginning of the century; and the figures given by Weber ($\frac{1}{8}$ to $\frac{1}{3}$ per cent. of all admissions into hospital) would probably fall short of those based on more recent statistics.

Bronchiectasis in its typical development affects both lungs; but it may be limited to a single lobe or even part of a lobe, or to one lung. The latter

* See "Clinical Journal," vol. ii., No. 1.

† See "Traité de l'Auscultation Médiate," 3rd edition, Paris 1831, vol. i., p. 205.

‡ See Lebert, "Klinik der Brust-krankheiten," Tübingen 1874, vol. i., p. 254.

condition, of which the following case presented a striking instance, we shall more especially consider; but I propose to touch upon the pathological features of the disease in general, contrasting it with Corrigan's cirrhosis, and to deal with the practical points of its diagnosis, prognosis and treatment.

THE CASE.

The patient, aged 62, a carpenter, of healthy parentage and antecedents, but a drinker of spirits, was admitted into St. George's Hospital, October 31, 1893 (No. 1889, Medical Register), and died on December 23, not of bronchiectasis, but of cancer, a circumstance to which I shall subsequently refer. He had been kindly put under my care by Dr. Rolleston, who diagnosed fibroid disease of the left lung, a diagnosis in which I concurred. But the patient sought hospital treatment less for his severe bronchial catarrh than for abdominal fulness and pain, shortness of breath, and progressive wasting and exhaustion. These symptoms were ascribed by me to ascites and hepatic cirrhosis; but the autopsy revealed omental disease, recognized by Dr. Lee Dickinson as columnar epithelioma, involving the peritoneal covering of the stomach, the cavity of which was reduced in size. The anorexia and almost daily vomiting, and the constant pain, were satisfactorily explained by these changes.

The history is briefly told. The patient had travelled in the Crimea, New Zealand, and Australia. Forty years ago he was laid up one week in the hospital at Balaklava with some chest affection. Every winter since then he has had a cough, which has been worse for the last twenty years. Two years ago he had another chest attack, lasting fourteen days, during which the expectoration was much increased (up to half a pint daily, the matter occasionally coming up in mouthfuls). He had also had pleurisy twenty-five years ago, on the right side. During the last six months he has lost fourteen pounds. His abdomen has been enlarged for two months (girth on admission, about thirty-four inches at the level of the navel), and he has had shooting pains at the hypogastrium. During the last twenty years he has had *hemoptysis* (about half a pint) occasionally. About ten days ago he vomited nearly a pint of dark fluid, like ink.

THE PHYSICAL EXAMINATION.

Dr. CYRIL OGLE's Examination, October 23rd.

Lies flat in bed; rather cyanosed. Nails incurved, with bulbous finger ends. Expectoration consists of masses of

green pus, not offensive. Pulse 98, regular, jerking, as of aortic regurgitation. Respirations 24; wheezing inspiration.

Heart: Apex-beat felt at the mid axillary line in the sixth space. First sound rather short. No murmur. Second sound clear, but faint. No diastolic murmur.

Lungs, anteriorly: Skodaic resonance rises high, and extends to within one inch of nipple level. The left side of the chest expands to a less extent than does the right; and the intercostal spaces are sucked in. At the left apex, a high-pitched percussion note, with cavernous breathing. Below the left clavicle to third space, a high-pitched note, with bronchial breathing and wheezing rhonchi. Below the third space, and over the entire axillary region the note is duller (more truly dull), with hard, gurgling râles, quite obscuring the breathing except in the axillary region proper, where it is tubular.

On the right side in front: Hyper-resonance everywhere, extending to one inch to the left of the sternum. No dullness. Rhonchi everywhere, with prolonged expiration.

Behind, right side: Wheezing rhonchi and prolonged expiration.

Left side: Bad percussion note, of high-pitched quality everywhere; hard râles everywhere, of large size (at base gurgling); in lower half of lung there is tubular breathing with increased fremitus and bronchophony.

Abdomen: Much enlarged. Fluctuation and dullness in flanks, changing with posture to resonance.

Liver dullness extends for three inches downwards from the upper border of the sixth rib.

Dr. EWART's Examination, October 28th.

Chest of large size, deep antero-posteriorly. Right mammary region more prominent than left. The left anterior and axillary regions seem flattened and move less than the right. The supraclavicular fossæ are deep, especially on the right side. Sternomastoids tense. Jugular veins pulsate slightly during expiration and are emptied by inspiration.

Percussion. Front, right side: Supraclavicular region resonant; clavicular resonant; infraclavicular less resonant with short vibration. Rest of chest resonant.

Left side: Supraclavicular region resonant; clavicular resonant with high-pitched note; infraclavicular slightly tympanitic. Second space resonant with short vibration. Third and fourth spaces resonant. Cardiac dullness absolute between the left nipple and a point distant 1½ inches from the middle line. (See diagram.)

Right axillary region resonant to base.

Left " " resonant down to the nipple level, below which the dullness is continuous with that of the heart.

Back, right side: Supraspinous fossa resonant, also remainder of lung to base.

Left side: Supraspinous fossa dull. Upper interscapular region imperfectly resonant. Lower interscapular region dull. Outer base dull, with tympanitic overtone (? stomach). Scapular region dull.

N.B.—The left intercostal spaces recede markedly with inspiration.

Auscultation. Right front: Breathing puerile, harsh, laboured, with indistinct tendency to râles and medium sized rhonchi.

Left front: Whispering pectoriloquy is loudly heard down to the second rib; less loud at the upper parasternal half. Below this the parasternal region gives harsh respiratory sounds, with hoarse, large râles. Over the region of pectoriloquy the re-

spiration is of loud cavernous, slightly amphoric quality; and there are rather large creaking râles; but none very fluid. The same kind of râle, of varying size, is audible over the whole upper part of the chest; but at the lower left parasternal region there are abundant, moist, medium-sized râles on cough.

Left axillary region: Loud whispering pectoriloquy over the upper two thirds, with cavernous respiratory sounds. The whole region down to the base gives abundant, rather large, inspiratory and expiratory râles (moist and viscid). Together with these râles is heard, down to the base, a loud creaking, croaking, and nasal cavernous breathing. These latter sounds ex-

THE SPECIMEN.

Two remarkable facts strike us at a glance:—(1) the atrophy of the left lung, which is barely visible from the front; (2) the extraordinary enlargement of the right lung, which not only extends beyond the middle line, but occupies nearly the entire upper front of the left chest, pushing before it the sternal fold of the right pleura. The thoracic contents as they appeared after removal of the

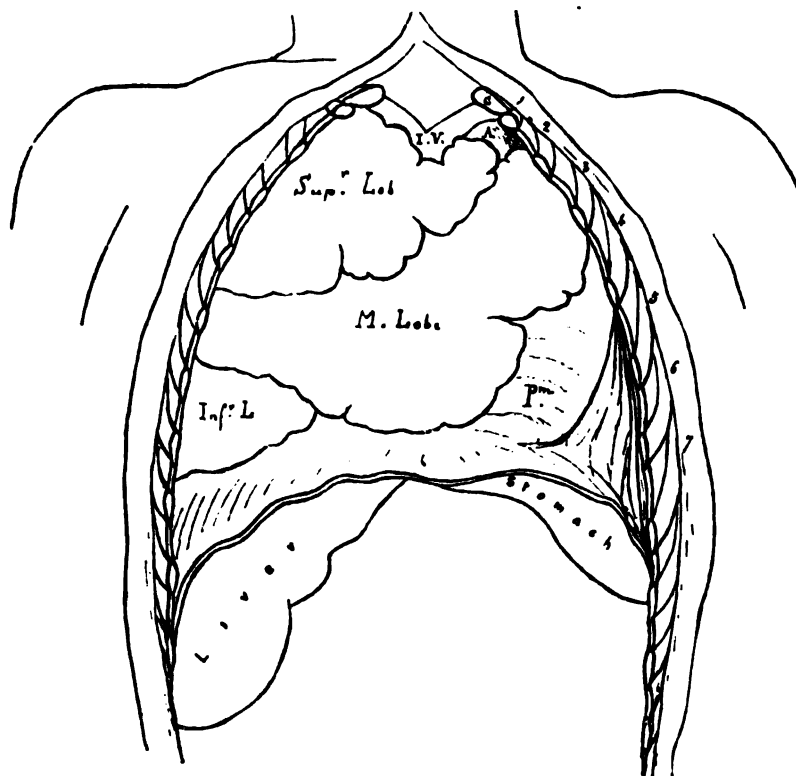


FIG. 1. The Thoracic organs *in situ*, seen from the front.

I.V. Right and left Innominate Vein.

A. A small surface of the ascending portion of the arch of the Aorta.

Pm. Pericardium, showing the attachment of the membrane to the left 4th chondro-costal junction, taking the place of the normal attachment to the sternum.

(Outlined from a Drawing by Dr. G. H. GOLDSMITH.)

tend over nearly the whole back, except in the upper interscapular and inner supraspinous regions, where the abnormal sounds are less loud. Their intensity increases over the dull patch along the inner border and middle third of the scapula. Here croaking and suction sounds are intense. Throughout the left lung neither vesicular respiratory sounds nor any fine crepitations are audible.

Voice sounds: The intensity of the voice sounds is not in proportion to the loudness of the cavernous breathing. The voice sounds have, throughout, a more buzzing character than usually belongs to pectoriloquy. The supraclavicular and infraclavicular regions give louder voice sounds than on the right side.

sternum and costal cartilages, are shown in the diagram. Nothing was visible except the right lung, which extended like a breastplate across the chest, the concavity of its upper margin exposing to view the two innominate veins and a small surface of the ascending aorta (the latter much farther to the left than normal), whilst its lower oblique border covered the greater part of the pericardial surface except the outer third, which remained exposed far to the left. The left lung was not to be seen.

There was *no anterior mediastinum* in the usual sense of the word, and no sternal attachment of the pericardium, the right costal pleura extending as a smooth layer behind the sternum and the left costal cartilages. The only connection between the pericardial surface and the anterior chest wall occurred at the fourth left costo-chondral junction (see diagram).

The lobes of the right lung were much altered in configuration, except the lower lobe, which kept its position, being to some extent adherent to the chest wall. The sternal edge of both the upper and the middle lobe, instead of being vertical, was rotated up towards the left, so as to become horizontal, and finally to assume a rising direction. The lower corner of the middle lobe was thus carried up as far as a point corresponding to the middle of the left clavicle. This strange displacement was partly due to two bands of adhesion shown in the small diagram.

Pleural adhesions existed at the right base. The upper and anterior surfaces of the right lung were perfectly smooth; but on reflecting the two upper lobes towards the right, they were seen to be connected with the aorta and pericardium respectively. The left lung was universally adherent. Pigmented fibrosis occurred at the apex; but the pleural thickening elsewhere was of a dense areolar, rather than of a fibrous type.

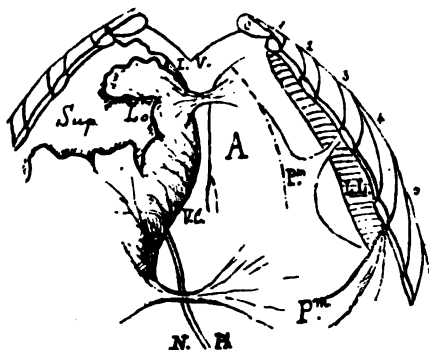


FIG. 2. Appearances displayed on reflecting the sternal portion of the right upper and middle lobes (seen from the right side).

L.L. The atrophied left lung, under cover of the left ribs.

A. The Ascending Aorta.

V.C. The Vena Cava.

Pm. Pericardium, unopened. The posterior surface of the right upper lobe is connected by a band to the Ascending Aorta; that of the middle lobe, to the pericardial surface.

N.Ph. Phrenic Nerve.

(Outlined from a Drawing by Dr. G. H. GOLDSMITH.)

The heart on reflecting the middle lobe was seen

to be dragged to the left. A band of adhesions extended from the right surface of the pericardium just posterior to the right phrenic nerve, to a corresponding point on the posterior surface of this lobe.

The pulmonary substance of the right lung was rarefied and light, the organ becoming much reduced in size after its removal from the thorax. The left lung was not only greatly reduced in size, but much condensed, the interval between the bronchial dilatations being occupied by coarse tissue with little trace of alveolation, and verging on fibrosis, although nowhere densely fibrous. The general colouration was pinkish-grey, black pigmentation occurring in excess at the apex, in scattered islands in the middle third, but being entirely absent at the base. *The infra-tracheal glands* were only slightly enlarged.

The bronchial system. There was no obstruction or scar in the trachea or main bronchus. The mucous membrane was thick and dusky red. Throughout the left lung occurred more or less spherical pouches, about half an inch in diameter, a few of them slightly larger. None of them were ulcerated. Their mucous membrane was thin and fibrous. Two or three pouches at the internal surface of the lung were almost free, the surrounding pulmonary tissue having suffered complete atrophy. The contents of the sacculations were muco-purulent, but not offensive. During life an examination had kindly been made by Dr. Rolleston, who could detect no bacilli of tubercle.

In conclusion, let me point out that the anatomical condition entirely bore out the results of the clinical examination, and in particular the extraordinary circumstance that respiratory sounds and râles had been observed *between the left edge of the sternum and the line of absolute cardiac dulness*.

THE CLINICAL FEATURES OF BRONCHIECTASIS.

The clinical features presented by the disease in its full development may be described as *general* and *local*.

I. THE GENERAL FEATURES.

Patient's aspect and general condition. Great importance attaches to these in view of the constantly recurring difficulty of diagnosis. The danger is to mistake bronchiectasis for phthisis. The diagnosis between them may be said to turn upon

a recognition of the characteristic aspect,—coupled with the behaviour of the temperature and respiration, both of which are about normal in the absence of complications. The bronchiectatic person differs from the phthisical in three essential particulars, which are correlated with the fact that the disease is one of progressive pulmonary impairment and of long-continued catarrh *without any grave constitutional factor*. In phthisis or in malignant affections the pulmonary disease infects the system through the blood or the lymphatics. Here it remains strictly pulmonary. Hence, the patient suffers mainly from respiratory inadequacy, and from the constant drain of an excessive mucous flow; and, putting aside the complications of fetid decomposition and of septic infection, he is not cachectic. Therefore

- (1) there is little or no emaciation; and,
- (2) anæmia is absent or slight,—although the complexion is usually sallow or muddy. Moreover,
- (3) the patient presents a more or less *venous type*, marked by full, dusky red lips and rather large veins, especially of the extremities.

To review these characteristics more in detail: you will notice that in sufferers from bronchiectasis

- (1) the orbits and cheeks are not hollow; the eyes are not large and sunken; the features are not sharp, as in advanced phthisis,—indeed, rather blunt than otherwise. The skin is not attenuated and tightly drawn over bony prominences, but of normal thickness and elasticity.
- (2) There is none of the waxy pallor and transparency of complexion, allowing thin blue veins to be seen. The veins are apt to show up in bronchiectasis, not because the skin is excessively transparent, but because the veins are turgid.
- (3) *The venous type varies in degree*: it is sometimes very striking. In this connection it may be observed that the tissues in bronchiectasis are not, as in phthisis, drained of their fluids by a process of ulcerative suppuration. We are dealing here merely with a physiological function, that of bronchial secretion, developed to an excess, and making, as it were, a physiological demand on the blood, the volume of which is kept up in proportion to the demand. But,

owing to the increased resistance offered to the pulmonary circulation, much of the blood is delayed in the veins.

The clubbing of the finger ends is extreme. This is another instance of the same venous influence. The small veins of the extremities are dilated,—a condition observed in various forms of obstruction to the pulmonary circulation, but more particularly in association with fibrous changes. For this peculiarity it is difficult to find an explanation. It would almost seem as though the venules of the finger ends dilated vicariously, instead of, or with, the right heart. In emphysema the fingers are not specially clubbed, but the right heart dilates. And it may be pointed out that in that disease the bronchial vessels, which, you are aware, establish a link between the pulmonary and the systemic circuit, are not so largely involved in atrophy. In phthisis, where the smaller bronchial vessels as well as the pulmonary capillaries are largely destroyed, there is no dilatation of the right heart, and the fingers are clubbed. In Corrigan's cirrhosis, and especially in bronchiectasis, the small bronchial vessels are destroyed on a large scale, and the clubbing is great, but especially great in bronchiectasis: in the latter, however, owing to the vascular fulness previously described, some dilatation of the right heart is apt to occur after a period of hypertrophy.

II. THE LOCAL PHYSICAL SIGNS IN BRONCHIECTASIS.

The thorax in bronchiectasis is not markedly deformed; and even in unilateral bronchiectasis there is not much loss of symmetry. This is a point of great contrast with Corrigan's cirrhosis.

Percussion. In spite of the extent of the lesions, and of the almost invariable existence of pleural adhesions, dulness is usually not absolute, as it is in phthisis at the seat of excavation. The great resonance found, in the case which has been described, at the upper part of the left chest, was in this instance due to the hypertrophied right lung. But it is not unusual for the affected lung also to be in parts emphysematous; and even the cavities within it may yield an imperfectly dull note owing to some remaining degree of aëration of their surroundings.

Auscultation detects the presence of *multiple areas of cavernous breathing*. And here a great distinction occurs between the two diseases. The cavities of bronchiectasis will be found not only at

the apex, but even more commonly in other situations in the lung, which do not in phthisis suffer excavation except at a very late stage. The character of the *cavernous breathing* differs from that commonly given by phthisical vomicae; it is usually less intense and somewhat veiled, frequently complicated with sounds of catarrh occurring in adjoining portions of the lung. Again the *bronchophony* is less intense, the thick mucous membrane and its viscid secretion probably acting as a damper to the vocal vibrations. It is most unusual for a bronchiectatic cavity to be absolutely dry; the dry cavernous breathing so often met with in phthisis will therefore not be found. *The adventitious sounds*, which are almost always present, are such as might be inferred from the presence of so much mucus. The catarrh extends to the whole lung, but the râles special to the sacculations are very characteristic. They are less metallic, more viscid than in phthisis, and resemble the suction, or valve-sound ("bruit de soupape"). In most cases the gurgling of a bronchiectatic cavity does not correspond to one sustained note, as in the common vomica, but is rounded off into a chromatic rise. The effect produced on the ear by the nasal, harsh, quacking sound, followed by a series of overtones, is excellently expressed by the word "croaking." The mechanism of this peculiar sound may be partly due to the tenacious character of the mucus, and perhaps also to the infundibuliform shape of the cavities, and to the more yielding character of their walls.

These few practical points by no means cover the ground of the physical signs of bronchiectasis, but they are those most likely to prove of service to you in doubtful cases; and with due attention to them, and to the clinical history, you will seldom fail to diagnose successfully bronchiectasis from phthisis.

(To be concluded.)

Whooping Cough.—Bromoform has again been recommended for this very troublesome complaint. It may be given in spirituous solution or on a lump of sugar. The best dose is one drop for each year of age of the patient (if a child) three or four times daily. The dose may be increased cautiously if the attacks do not diminish in the first three days. The drug is only slightly soluble in water. It is said to check the vomiting, so improving the appetite and curing the disease in three weeks.—*Pellicier*, 1893.

A CLINICAL LECTURE ON SOME CASES OF RETENTION OF URINE FOLLOWING OPERATION.

Delivered at the London Hospital. Oct., 1893,

By O. MANSELL MOULLIN, F.R.O.S.,

Surgeon to the Hospital.

GENTLEMEN,—Last week you saw me operate upon three patients who were suffering from varicocele, and two with hæmorrhoids. The former were young, the latter middle-aged; all were perfectly healthy, so far as their urinary organs were concerned; none of them had suffered from stricture or enlargement of the prostate. By a somewhat unusual coincidence retention of urine occurred in all of them. In three a catheter was only required once or twice; one did not regain power over his bladder for three days; and the fifth, a man 43 years of age, who had been suffering from external hæmorrhoids, was for more than a week in this condition. His bladder seemed to have passed into a state of complete atony, for when a catheter was introduced, the urine simply passed away through it, the stream rising and falling with respiration; and it is only now, ten days after the operation, that it has begun to recover its power.

The same thing is, of course, of common occurrence after accidents and injuries, especially railway accidents. In old people, the strength of whose nervous system and muscular mechanism is not very great, the most trivial hurt will cause it; in younger ones the injury or operation must usually be a severe one, unless it involves the pelvis or the pelvic organs, when very little is sufficient; in children it is, comparatively speaking, rare. It is often spoken of as an example of retention of urine caused by spasm. Without denying that this happens sometimes, I need not tell you that in the vast majority of the cases that I have mentioned, if not in all, the retention is simply due to shock.

To understand what takes place under these circumstances it is necessary to consider briefly the mechanism of micturition, and especially the question of the existence and locality of the sphincter of the bladder, which is supposed to be in a state of spasmodic contraction and to prevent the exit of the urine.

The prostatic portion of the urethra and the bladder are developed, as you know, from one and the same structure, the allantois, and have no genetic connection with the rest of the urethra, which is developed in an entirely different way, and which, in one class of mammals, never joins with the rest, but remains throughout life separate and distinct. There is no sphincter at the neck of the bladder shutting it off from the urethra. This was shown long ago by Guthrie; and although Ellis, and, more recently, Harrison, have described one, there can be no question that Guthrie was right. I have searched for it on many occasions and have never succeeded in finding it. The circular fibres are, I admit, a little closer together at the neck than they are over the fundus, and cease, or almost cease, for some little distance below; but there is nothing in this that deserves the name of a sphincter; the increased thickness is very slight and very gradual; nothing like a band is formed; and the fact that the prostate supports the walls below is sufficient reason why the muscular fibres are so few. The true sphincter is lower down at the apex of the prostate, where there is a considerable collection of unstriated muscular fibre, and where there is, in addition, outside and separate from the walls of the urethra, a strong voluntary sphincter known as Henle's muscle.

This begins with a few cross fibres on the anterior surface of the prostate, about the middle of its length, and growing stronger and larger as it descends, forms at last a compact layer, surrounding the urethra and emerging into the compressor urethræ below. It is this that forms the true sphincter.

The best physiological evidence of this is the way in which micturition is carried out in infancy. At that time of life it is purely a question of a reflex act. The urine, as it flows into the bladder, does not collect in a passive receptacle; the muscular coat of the bladder maintains throughout a tonic contraction upon its contents, dependent upon the lumbar spinal cord. As it gets fuller the tonic contraction of its walls at length overcomes the elastic resistance and the tonic contraction at the orifice, a drop of urine passes through; at once a message is carried up to the spinal cord; the prostatic portion of the urethra relaxes and the bladder contracts. In infants this takes place at intervals, regulated by the quantity and the quality of the urine. About the end of

the first year of life, when the paths down which voluntary impulses travel have become developed, the striped sphincter of the urethra comes into play in exactly the same manner, and at very much the same time, that the striped sphincter does in the neighbouring bowel, as a result of education. Now, when a similar message is conveyed to the spinal centre, the will decides whether micturition shall take place or not. In this matter there is a very close parallel between the bladder and the rectum. Each is composed of unstriated muscular fibre, which is somewhat thickened at the exit, more so in the case of the bowel than in that of the bladder. Each is guarded by a striped sphincter, the use of which has to be learnt as part of education. In each the most sensitive portion of mucous membrane is that which lies just inside the thickened fibres; a stimulus applied to this spot in either organ immediately calls into play vigorous muscular contraction in the part above; and in each the first step that follows stimulation is the relaxation of the unstriated muscular fibre around the outlet. In the male the question is complicated by the addition to the urinary organs of others that are concerned partly or wholly with the sexual function, the penile portion of the urethra, for example, and the prostate; if the anatomy and physiology of the urethra in the female are taken into consideration the parallel is more easily seen.

There is one point, however, in which they differ, and that is the relative strength of the voluntary sphincter and of the involuntary muscle above. The sphincter in the bowel has only limited power; after a time it must give way. In the case of the bladder, so long as the mucous membrane is healthy, the sphincter is the stronger of the two, and retention of urine may voluntarily be carried so far that the power of expulsion is lost; but this only holds good under normal conditions; when there is any inflammation at the neck of the bladder, or when there is the irritation of a calculus or a new growth, the stimulus becomes so strong that the voluntary sphincter is at length compelled to give way, just as it is in the bowel.

The prostatic portion of the urethra, when micturition is beginning, is dilated and becomes physiologically, as it is genetically, part of the bladder. The bladder when empty is somewhat triangular in shape. As it fills, the upper wall is raised, and the angles are rounded off until it becomes more or less spherical, the urethra being

closed. When it is full, and the desire to urinate is present, the prostatic portion of the urethra dilates, and the cavity becomes pear-shaped. This can be shown by a very simple experiment. If a catheter is introduced into the bladder when it is full, but before there is any desire to urinate, until the urine begins to escape, and then if the same thing is done with the same subject when the desire is present, and the length of the catheter required is noted on each occasion, it will be found that there is a difference of more than half an inch between the two. The urethra is shortened by so much as soon as the desire to micturate is felt.

In cases in which retention of urine follows injuries or operations it is probable that the shock involves the kidneys as well as the bladder, and that very little urine is secreted in the first few hours. At any rate, patients can go on for a much longer time than is usual without any wish to empty the bladder, whether this arises from the small quantity secreted or from inactivity and dulness of the central nervous system. Then by degrees, if relief is not given, the bladder becomes more and more distended, until the elastic resistance of the walls becomes greater than that of the orifice, and the urine enters the prostatic urethra. Under normal circumstances contraction of the detrusor would follow, the prostatic portion of the urethra would dilate, and the result would depend upon the orders sent down to Henle's muscle and the compressor urethræ. As matters stand the lumbar spinal centre is inhibited by the shock it has sustained, and for the time being is unable to act for itself, or to transmit orders sent down to it from the parts above. The walls of the bladder have lost their tonic contraction; the prostatic portion of the urethra cannot dilate, for the muscular relaxation upon which this dilatation depends (indeed, it may be expansion and not merely dilatation), is as much under the control of the nerve centres as muscular contraction; and the voluntary sphincter cannot act, for the impulses that should travel to it through the lumbar centre are blocked. As a consequence, the urine trickles down the urethra as soon as the walls of the bladder are stretched, past the voluntary sphincter and past the unstriated muscular fibre that lies inside it, and overflow follows, the bladder remaining distended to its utmost.

Provided the urinary organs are healthy, there is never any difficulty in passing a catheter in these cases; a full-sized one and the softest that

can be found should be selected: there is never any spasm and it is never gripped by the muscular fibre of the urethra. The number of times it will be required may be gathered by observing the manner in which the stream flows out. If it comes out forcibly as it should do through a full-sized catheter from a distended bladder, it is evident that the detrusor is recovering. If on the other hand, it flows away gently and quietly with a rise and fall synchronous with respiration, it will be required again in a few hours time, and very likely for several days.

Great care must be exercised in all these cases to avoid anything that approaches extreme distension. The patient may not complain of it, but it may, for all that, inflict irreparable harm upon the bladder. The tone of its muscular walls is temporarily in abeyance, owing to the shock the nerve centre in the lumbar spinal cord has sustained. If when this is beginning to return the walls are allowed to become over-stretched, permanent atony may follow. Unstriated muscular fibre that is stretched for any length of time beyond a certain point, sometimes loses its power of contracting. The loss of tone that arises from shock, I believe, always passes off, although, as in one of these cases, it may take some considerable time. Atony from over-distension, on the other hand, is occasionally permanent, even in young men. There are not a few instances in which retention of urine, voluntary at first, has been so prolonged that at length the muscular coat of the bladder has lost its power of contracting, and has never regained it. The absolute necessity for the regular periodic use of a catheter in these cases must always be borne in mind. Retention arising from other causes can sometimes be relieved by hot baths, or in other ways; here nothing but catheterization is of any use. In women, especially young women, it is allowable to wait longer than in men. The bladder in them seems more tolerant of distension, and the mechanical resistance in front is much less; moreover it sometimes happens that if the use of a catheter is commenced in them, it is a little difficult, and requires great judgment to dispense with it again. Fortunately, the occasion does not arise so frequently in them as in the opposite sex.

It is impossible to predict beforehand whether any given patient will or will not suffer from this troublesome complication. I believe it is, as I have said already, more likely to happen in the old than in the young, in the male than in the female,

but I cannot lay down any general rule. The strongest and most active may suffer from it. I have indeed sometimes thought that they were more likely to than those who had been confined to bed for some time before. Therefore you must always warn your patients of the possibility of its being necessary; for I need not tell you that the passage of a catheter is very reasonably regarded by them as a painful and a serious matter. I believe the risk of its occurring may be very greatly diminished by placing them for a week beforehand on small doses of Strychnia, and this may be continued with advantage after the operation. If, however, it has not been given them before the operation, and retention does occur, I think it is better to leave the nerve-centres alone, and let them rest until the inhibitory influence of the shock has passed off, than to stimulate them in their weakened state.

A CLINICAL LECTURE ON ACNE.

Delivered at University College Hospital, Jan. 13, 1894, by
H. RADOLIFFE CROCKER, M.D., F.R.C.P.,
Physician to the Hospital.

I PROPOSE this morning, Gentlemen, to give a short lecture on acne. There are several affections of totally different nature included under this term. I propose, first of all, to show you what the different affections are that have had that name, and then to go more particularly into the treatment of the commonest form of it. There are also a great many, which may be called artificial varieties of acne; these I will only indicate to you, in order that you may not trouble yourselves by attending too much to them.

The primary and chief forms of acne are acne vulgaris, acne rosacea, and acne varioliformis. We have here two cases, one an extreme example of acne vulgaris that has improved by treatment, the other of acne rosacea; and I show you here a drawing of acne varioliformis, which is a very much rarer disease. We shall first of all take the two varieties that we do not intend to discuss very fully.

The rarest form, acne varioliformis, is essentially a disease that attacks the border of the scalp; and not only that, but goes back into the scalp. This is a very important point, because acne vulgaris and acne rosacea do not affect the hairy parts. Therefore, if you see a scar-leaving, acnei-

form eruption affecting the forehead and temples, and going back into the hair, you may be pretty sure it is acne varioliformis. There is a syphilide which somewhat closely resembles it, but I shall not go into that now. Although this is the most characteristic position in which acne varioliformis occurs, it is not always limited to it. By repeated attacks, for it is a very chronic and recurrent disease, it may spread all over the face, and in exceptional cases we may also see it on the trunk, both front and back; but still in the great bulk of cases it is limited to this position, so much so that it has been called acne frontalis. Various other names have been given to it; but the name by which it is best known, and which is most distinctive, is acne varioliformis, *i.e.*, small-pox-like acne—the adjective being applied on account of the pitting after the disease has healed, which is exactly like that of small-pox. I regret we have not a patient present to show you; there are several attending the hospital, but none of them happen to have come to-day.

Acne rosacea is a very common disease; acne varioliformis, we saw, was an uncommon condition. Here, in this woman, we have a living example of acne rosacea; while in this drawing we have represented a typical case, showing the full distribution of the condition. It occupies the middle two-thirds of the face, seldom encroaching beyond that. Outside of that area this woman is quite free from the condition. As in this case, the forehead often escapes. When it affects the forehead it is, for the most part, confined to the centre, and the sides of the face are perfectly clear. On the other hand, in acne vulgaris the eruption is, as a rule, most developed upon the sides of the face. The distribution is thus, you will see, of the greatest importance. This distribution on the prominence of the cheeks and the chin is not exclusively the appanage of acne rosacea, though an acne limited to this position is sure to be of this form. Here is the representation of a disease, adenoma sebaceum, in which the distribution is very much the same. Acne rosacea is a disease due to disorders of the stomach producing dyspepsia and flushing of the face, or, in women, it may be due to uterine derangement; but even in cases where there is uterine derangement there is frequently associated dyspeptic trouble also; so that we might really call acne rosacea the dyspeptic acne.

Acne vulgaris is, for the most part, a disease of youth; it is not absolutely and exclusively so, but

you generally see it from puberty to the age of 25 or 30. You may say roughly that acne vulgaris occurs from 15 to 30. There are a few cases that may go on longer, especially when the back is affected, but as a rule that is their limit. On the other hand, acne rosacea generally begins from 30 and onwards—just when the other leaves off. The reason for the occurrence of acne rosacea at this particular period simply is that up to that time most people's digestion holds out.

Acne vulgaris, then, is a disease of puberty; and we must remember what changes occur then. At that time all the glands take on an increased activity; especially is this the case with the sebaceous glands. When that occurs, very often the physiological limit is slightly overstepped, and we get pathological activity. This slight overstepping of the physiological limit often produces the condition which we call comedo. The comedo is a little black papule in the skin due to a blocking of sebaceous matter plus a little dirt or some actual colouring matter,—which of the two is not of importance,—it is really a sebaceous plug. The probability is that there is increased formation of sebaceous matter, and that it does not undergo to the full extent the fatty degeneration that usually occurs, so that the fat cannot escape, but blocks the gland. Now, anything that blocks the orifice of a follicle is very likely to lead to inflammation of the follicle. We see that not only in this condition, but also amongst the workers in tar, paraffin, and other things that plug up the follicles. They get a form of acne as a consequence; and so we hear of "tar acne," "paraffin acne," and other forms due to the nature of the occupation. They are all, however, really the result of the mechanical plugging.

When this occurs, inflammation supervenes round the gland with a suppurative point,—the condition called the common acne pustule. In the great majority of cases they are of moderate size when superficial; but sometimes they are formed deep in the tissues, and then you get hard nodules, which have been called acne indurata: they are commonly, however, only an indication of stage. The young man before you shows an additional feature. The inflammation has spread beyond some of the sebaceous glands into the subcutaneous tissues, so that there have formed what we may call acne abscesses. Of course, in one sense, every acne pustule is a small abscess; but this man had abscesses, some of them as big as a hazel nut, one or two as large as a walnut.

These have been opened and syringed out, and you may see the remains of them now. The point about these indurated acne pustules is that even when there is only a red papule there is always pus within them; and that is of importance, because you need never wait until you get a superficial pustule before you puncture it; and the sooner it is punctured the less likelihood there is of a scar being left.

This man illustrates the different forms. Here is a comedo; here is a superficial pustule, "acne simplex"; here are deeper pustules, "acne indurata"; and here are the remains of comparatively large abscesses. The eruption, of course, occupies mostly the sides of the face; but, this being an extreme case, it is by no means confined to it. The commonest position for this disease is about the face and the sides of the neck adjacent; but it also occurs on the body, especially the back and chest, but not so much the chest as the back. In extreme cases, the back may be covered with these pustules, or the remains of former ones, and at all stages. Some people's backs are one mass of scars and stains, and, of course, more recent lesions when the process has gone on for years. These are the cases that have not been limited by the age of 30, persisting, perhaps, to the age of 50 or more.

In still more exceptional cases you may get a generalized acne, the whole of the follicles of the body undergoing an inflammation. It occurs chiefly in persons who have undergone some great depression of health, especially from starvation, but any other cachectic condition may excite it, and then you get a general folliculitis which has been called "acne cachecticorum." A variety of this occurs in young children—the "acne scrofulosa." The same distribution seems to govern nearly all cases of the latter, namely, the extensor aspect of the limbs, but especially the region of the buttocks and the lower part of the back. In nearly all these cases, which are rather rare, other scrofulous manifestations are very evident,—glandular enlargements, suppurations elsewhere, perhaps bone disease, etc. These are the most extreme developments, and are seen in quite young children.

With regard to the treatment of acne vulgaris, one great point is to ascertain the condition of the alimentary canal. A large proportion of them have constipation, or some other disorder of digestion. As a consequence, they are very often very anæmic also, and that condition may require treatment; but

in a great many of these anæmic cases even the anæmia is due to fecal accumulation; and it is in that direction you must address your efforts. If you give Iron to these cases simply on account of their anæmia you rather aggravate than improve their condition. The first thing to do is to treat the alimentary canal and then to give your Iron. Sometimes you can combine the two. A favourite mixture of the late Mr. Startin, who was very clever at composing what I might call therapeutical blunderbusses, consisted of Sulphate of Iron, Sulphate of Magnesia, dilute Sulphuric Acid, with flavorants and carminatives to prevent griping. That, no doubt, was a very valuable mixture for many cases of acne with constipation. If, however, the tongue is much furred and shows prominent papillæ, I think it is better to begin with alkalies and laxatives such as

Sod. Bicarb.	gr. x
Ext. Cascar. Sagrad. Liq.	...	℥ x-xx	
(according to the amount of constipation present)			
Tinc. Nuc. Vom.	...	℥ vii-x	
Aq. Menth. Pip.	ad	3j	
(or any other carminative).			

That kind of mixture suits a very considerable proportion of cases. After they have taken this for a week or ten days, if there is any indication for Iron you can give it in the shape of Reduced Iron pill, two to three grains after dinner or oftener, if you think it desirable. You thus remove the dyspeptic conditions, and treat the gastro-intestinal catarrh which is so often present; and the small amount of Iron given in this way, which is quite enough to get the therapeutic effect, does not seem to upset them so much as when larger doses in the form of the sulphate are given. Of course this is not done in a routine sort of way. You must examine the patient, and find out what defects there may be either in themselves or in their circumstances. Do everything you can to invigorate the patient. Cold sponging in the morning, active exercise, plenty of friction to the skin, regulation of diet—all these things must be attended to. As a rule, all things that are apt to promote fermentation should be avoided. Malt liquors, effervescing wines, sweets, and pastry are to be shunned almost invariably. Occasionally, however, you have very little in that way to treat; but in any case the diet should be as simple as possible, so as to give the stomach as little trouble as may be.

With regard to the local treatment, the one thing that we want to do is to remove these little black plugs. There are many ways of attempting

this, and it is not quite so easy as it appears. One thing to do as a preliminary is to soften the secretion by means of steam, or by bathing with very hot water, and firm friction. After that various instruments with a central hole may be used, and almost every dermatologist has invented one. I have had one contrived, but do not attach particular importance to that. One of the favourite forms is a little circular cup with a central hole; this is set in a handle. The hole may be of various sizes. One of these was invented by Mr. Clover, who, a few years ago, was well known as an anæsthetist, and a very ingenious man in many ways. This Clover's acne-presser has been re-invented repeatedly with various curves. His was set straight in a handle convenient to hold; but one gentleman has put a curve to it and called it his acne-presser, and another gentleman has had one made with the cup at a right angle to the shaft.

Another very effectual plan is the good old-fashioned one of using a watch key; but the drawback to its use is that it has sharp edges, which are apt to excoriate the skin unless it is carefully used. Modifications of that have, therefore, been made in the form of silver tubes with a slightly rounded edge.

With all these things, however, it is better to have a preliminary softening application, either of steam, hot water, or certain soaps. One of the best of these consists of equal parts of soft soap and Spirits of Wine. A piece of flannel is dipped into warm water, and then into this liquid soap application, which is rubbed firmly on where the black specks are most abundant. After that the acne-presser may be used with advantage. If the soap produces a local irritation, as it is apt to do after a few days, it is better to suspend its use or reduce it to every alternate day, or every third day, instead of daily, in the meantime applying an application that will reduce the hyperæmia. For this purpose various Zinc or Bismuth applications are useful, with a smaller amount of Perchloride of Mercury, such as:

Calamine	3ij
Oxide of Zinc	3ss
Perchloride of Mercury	gr. 1
Glycerine	℥ x
Rose Water	3j

Another plan much favoured in Germany and America, is to scrape the surface where the comedones are abundant with a curette. It no doubt removes many of the sebaceous plugs and shortens the treatment somewhat, but produces temporary

disfigurement, making the face red and sore for a few days. Some apply slightly caustic alkaline solutions, either of Potash or Ammonia, before curetting, but great caution is required in using them.

If you want to treat the acne, not simply to reduce the inflammation, Sulphur is one of the most useful applications. A favourite lotion is one composed of 2 drachms of Sublimed Sulphur; 2 of Spirits of Wine; 2 of Ether; and 2 of Glycerine, in Rose Water to 8 oz. This must be dabbed on freely during the day, the soap being used at night.

Where there is much pustulation, one of the most useful applications is one that Erasmus Wilson employed largely—a drachm of the Hypochlorite of Sulphur to an ounce of lard. This is to be rubbed into the pustules at night, and is a very valuable application. There is one caution about it, that it should always be freshly made, and care must be taken that the Hypochlorite of Sulphur is in the powder form and has not been exposed to the air, because on one occasion a lady for whom I had prescribed this, wrote that it had made her very much worse. Having perfect faith in what I had done, I wrote for the application. The moment I undid the parcel the stopper blew out. It had been made with a liquid. You can improvise Hypochlorite of Sulphur by using the direct action of acid on Sulphur. That, of course, undergoes very rapid decomposition; and it has excited a very considerable amount of local irritation. Be very careful, therefore, to order the powdered Hypochlorite, and see that it is fresh.

There is, however, a still more efficacious way of dealing with these pustules, and that is by puncturing each one as early as possible, and then syringing it out. For these pustules, like boils, are auto-infective. Although there is an underlying constitutional state in this, as in many instances, yet no doubt staphylococci get in and flourish more in some people than others. If you disinfect each of these little abscesses, you will exhaust the supply much quicker than if you treated the condition by internal remedies alone, or with such applications as I have mentioned. A narrow-pointed knife should be passed in until you reach the pus—for there is always pus there, and the sooner that is evacuated the better. The pustule is then syringed out with a hypodermic syringe, containing a 1 to 60 solution of Carbolic Acid, or a solution of Perchloride. I cut off the pointed end of the hypodermic needle, making it simply a rounded tube. If there are a large number of pustules to do,

I puncture some two or three times a week. After a fortnight or so you will find it perhaps only wants to be done once a week. The deeper ones having been got rid of, the patients may be instructed to open the more superficial ones for themselves. The difficulty to be encountered in this treatment, is that people don't realize that as soon as there is a little tender nodule in the skin, there is pus there, and that the sooner it is let out the better. Of course, as a rule people are not very eager for the knife in any form, considering it in the light of an operation; but even ladies will consent to it if you can only convince them that scars will be prevented if each nodule is punctured early enough.

The indurations left from these abscesses are best treated with mercurial applications. Mercurial Plaster is the most convenient, or Unguentum Hydrarg. may be rubbed in. Unna has devised several plasters of this kind. I generally use either the Emplastrum Mercuriale of the German Pharmacopœia spread on linen, or the Emplastrum Vigo of the French—a very ancient remedy, and of the blunderbuss order, containing a vast number of ingredients. It has been easier to go on with the old formula than to decide which of its constituents are useless, and which the really valuable ones. This plaster is worn at night, and during the day one of the lotions is used, which partly conceal the eruption and partly diminish the hyperæmia. The Calamine lotion I gave you is very useful for this purpose. The great thing is to adapt the colour to the patient's complexion. Calamine is a native Carbonate of Zinc, which varies very much in colour, according to the amount of Oxide of Iron you use. It is better to get the pink varieties, and sometimes a little raw umber may be added; or sometimes I add a little Compound Tincture of Lavender, which is a red preparation, to get it a little more pink, and so match the complexion. It is painted on, and with the finger or a piece of wool is diffused over the skin, so that it is not conspicuous in any one part. It is then allowed to dry.

That is the principle on which one treats all these various forms of acne adolescentium. With general hygienic treatment, they always effect a considerable improvement in the patient. Although you cannot in the case of a boy or girl of 16 guarantee an absolute cure in a short time, you can always say that the amount of the disease shall be reduced to a few occasional pustules. As the patients get older and more healthily developed, the disease will be absolutely eradicated in most instances.

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WEDNESDAY, FEBRUARY 28, 1894.

A CLINICAL LECTURE ON RESECTION OF THE HEAD OF THE HUMERUS FOR LUXATION.

Delivered at St. Mary's Hospital, Feb. 2, 1894, by
EDMUND OWEN, M.B., F.R.C.S.,
Surgeon to the Hospital; Senior Surgeon to the Children's
Hospital, Great Ormond Street.

GENTLEMEN,—I wish to bring before your special notice to-day three cases of dislocation, in which I have recently had to resort to the operation of resection of the upper end of the humerus. The first is that of a woman of mature age, who had a neglected dislocation of the humerus below the genoid cavity. The second is that of an epileptic man who was worried by an oft-recurring subcoracoid luxation, and the third is that of a woman who had fracture through the anatomical neck of the humerus combined with a subcoracoid luxation.

Before entering into details, let me say, briefly, that the method of operating was the same in each instance—by a four-inch incision descending from beneath the clavicle midway between the acromion and coracoid processes. The incision passed a little to the outer side of the groove between the pectoralis major and deltoid, so as to avoid wounding the cephalic vein. It traversed the anterior fibres of the deltoid. The capsule of the joint having been opened, and the tendon of origin of the long head of biceps having been lifted out of its bed, the arm was rolled outwards and the subscapularis detached from the lesser tuberosity. It was then rolled inwards, and the supra- and infraspinatus and the teres minor were detached from the greater tuberosity. Then the end of the bone was thrust up through the wound, freed from attachments behind, and sawn off with a wide, oblong saw close below the tuberosities.

The wound was in each instance closed by a deep, continuous suture, except at the lowest part, where a drainage tube was left in for twenty-four hours.

The dressings consisted of wood-wool tissue, than which I know nothing more trustworthy or

convenient. In each case the wound healed promptly, and practically without any suppuration.

The temperature charts, as you will notice, were uneventful and uninteresting.

Resection for unreduced subglenoid dislocation.

The first case, then, to which I wish to direct your attention, is that of the woman whom I will shortly have called in. She admits to being 42 years of age; but, judging from her manner and appearance, she has in all probability omitted to reckon something more than a decade. It would be not only ill-mannered on our part, but probably useless to inquire too closely into this delicate matter; still, I must refer to the question of age, or rather of *agedness*, in connection with unrecognized and neglected dislocation of the humerus, because it may have a considerable and important influence on the treatment of such a case. Obviously, a surgeon is not justified in using as much force in attempting to reduce an old-standing dislocation in an aged subject as in one whose tissues are in a state of perfect nutrition, and, therefore, elastic and strong.

There are various elements of danger in connection with forcible attempts at the reduction of an old-standing luxation: The first that I mention, fracture of the upper part of the shaft of the humerus—probably at the surgical neck—is the least important of them, and the one which is most likely to occur. It happened, I well remember, in the case of a powerful, middle-aged man, whose unreduced humerus I was helping the late Mr. Haynes Walton to replace in private practice more than twenty years ago. In all such cases the upper extremity of the humerus becomes firmly fixed in its new position, and when, with his heel in the arm-pit, and his hands firmly grasping the elbow, a strong surgeon is doing his best to replace the head of the bone, a sudden adduction of the arm may easily cause the upper end of the shaft to break. For, acting in this manner, a tremendous leverage is obtained. If such an accident occurs, the surgeon must put up the limb as if for fracture, and, after due lapse of time, he should resect the upper extremity of the humerus.

Another element of danger in these cases is rupture of one of the axillary vessels. Irritated

and slightly inflamed in its bed, it is little wonder if the external coat of one of these vessels becomes glued to the surrounding tissues; and then, if it is roughly and severely pulled upon and stretched, it is more than likely that it will give way, and especially so if its coats have lost their elasticity in the course of senile change. Körte, of Berlin (*Lond. Med. Recorder*, 1889), had collected seventeen cases of this disaster.

Another accident that may happen—it has happened in recent years in a London hospital, though possibly the case was not recorded—is that the skin and soft parts surrounding the joint may give way under the pressure of the heel in the arm-pits, like so much wet brown paper, rendering the dislocation compound. Further, I believe that I am correct in saying that in at least one case the arm was actually pulled adrift from the trunk. Fortunately, the days are now past when a patient must be either subjected to some serious risk or left with a stiff, painful, and comparatively useless arm. The happy alternative has been found in the shape of resection of the end of the humerus.

This is the patient, Gentlemen, a married woman, 42 years of age. She was admitted on 13th December last. A little more than two months previously she had been pushed down by her husband, and in the fall she had dislocated her left humerus. Most likely in the act of falling she automatically thrust out her hand and arm to break the shock, fixing the arm by the energetic contraction of the pectoralis major, the latissimus dorsi, and of the other muscles which are inserted into the humerus. The escape of the head of the bone through the capsule, probably took place at the moment that the hand or fore-arm came in contact with the ground, and it was due partly to the sharp and sudden impact which the bone received at the fall, and partly to the sudden and violent contraction of the muscles. The head of the bone usually makes its escape through the lower part of the capsule, between the subscapularis and the teres minor.

A feature of considerable interest in this case was that, having escaped from the lower part of the capsule, the head of the humerus remained in this position, resting against the axillary border of the scapula. As a rule the head of the bone is carried up towards the coracoid process; but, as I have said, in this case it remained where it was, and constituted a subglenoid luxation. You will remember that a few weeks ago we worked out

this case together in the Manvers Ward. The chief features of the dislocation were that the elbow was well abducted from the side, and that the measurement from the tip of the acromion process to the external condyle of the humerus, was increased by over an inch; that the head of the humerus could be clearly made out by the fingers in the arm-pit, and that there was no fulness in the region of the coracoid process. These features sufficed to distinguish it from the sub-coracoid, which is the commonest form of dislocation. She may now leave the theatre, please.

The surgeon who saw the woman just after the fall, said that she had not only dislocated her shoulder (he fully recognized that lesion), but that she had also broken the surgical neck of the humerus. He, therefore, put up the arm on an angular splint. As a matter of fact, she had sustained no fracture, as was shown at the operation. And although I am not going to find fault with him for having diagnosed more than actually existed, still I think that he was not a little indiscreet in failing to examine so important a case under ether, which, it appears, he omitted to do. Nothing is easier than to commit an error of diagnosis in the case of an obscure injury to a shoulder joint, and this very fact should make us all extremely careful in our examination of such cases, and charitable to any of our brethren when we happen to detect an old and unrecognized dislocation in one of his patients. All doubtful or obscure cases should be placed under an anæsthetic, and the examination should be made conjointly with some surgical colleague.

The explanation of the surgeon having diagnosed fracture of the surgical neck of the bone as well as dislocation, was probably that in his examination he made the bare humerus rotate against the axillary border of the scapula. This would give a mistakable feeling of crepitus. The great point is that on rotating the shaft of the humerus in the case of fracture, the head does not rotate with the shaft. But the head of the humerus is in so comparatively an inaccessible situation, and is so cylindrical, that it is difficult in some cases, and especially so in stout women, to say for certain whether the head rotates or not. Altogether, then, the mistake was quite pardonable. At any rate, the dislocation was not overlooked. The surgeon apparently possessed that which the French cynic abjured—too much zeal. The woman has told us that she has been much more comfortable

since the operation; that she has lost all pain and discomfort from the arm, and that she is already beginning to use the arm with ease and success. The specimen which I now send round shows that the end of the bone was sawn off just below the tuberosities.



Before submitting this patient to operation, we had her in at Consultations, when it was agreed that under either a fair trial should be made to restore the bone, and that if this failed, resection should be resorted to. Having, therefore, failed to reduce the dislocation by Kocher's method, I cut down upon the deeply-lying head of the bone, and, having divided the small muscles, I set to work to lift it into the socket by a strong raspatory. But this proved no easy task. We found the muscles blood-stained and infiltrated. For a moment, after the bone was in place, I felt inclined to leave the muscles to attach themselves once more to the tuberosities, as in a case of a somewhat similar nature which Mr. Gould* successfully treated after that method. But in the face of all the disturbance to which the parts had been subjected, I thought it best to carry out the resection as originally agreed upon. And I think that the decision was right.

Resection for frequently recurring dislocation.

The second case is that of a man of 25 years, a gardener. He tells us that up to his sixteenth year he was quite well, but that at that time he was attacked with an epileptic fit. Since then he has been subject to fits, which overtake him at intervals, varying from three weeks to six months. He further tells us, and with great precision, that when he falls in one of these epileptic fits, he throws out his arms, and that with a "sudden jerk" he usually puts out his left shoulder. The first time that the humerus slipped out of the socket was about five years ago, and since then the

same thing has happened nearly twenty times. On each occasion he has had to seek professional assistance for the reduction, under chloroform, by his country doctor, and he tells us that not only is he tired of paying the very modest fee of a guinea on each occasion, but that the pain and weakness left in the arm after the reduction, render him unable to work for three weeks or a month.

Some of you may remember that the man was in the hospital under my care about two years ago, having been sent up by his doctor with a view to an operation being done to prevent recurrence of the luxation. One or two of my colleagues, however, thought that some alternative might be found to operation in the shape of a leather retentive apparatus. So we sent him home again that this might be tried. It was tried, and found to be ineffectual, dislocation recurring in spite of it.

On November 21st of last year, the man was prepared for operation, and, being under the anæsthetic, a little manipulation easily produced a subcoracoid dislocation, which could be as easily reduced. The head of the bone with the tuberosities was resected, and when this was done, the rent in the capsule by which the bone was accustomed to make its exit, was seen and recognized. The wound healed promptly, and at the beginning of the third week gentle movements of the limb were begun. You notice that he has now fair use in the arm, and all promises well for its future usefulness. For his work as a gardener the limb will serve him excellently, and it is quite



Head of Humerus, showing deep notch.

certain that he will have no recurrence of his old trouble, for the simple reason that there is no longer any humeral head to be luxated. In all probability a very useful false joint will form in due course.

Here is the piece of bone removed. You notice

* "Trans. Med. Soc.," 1892.

that it consists of the articular surface and the tuberosities. I thought it better to remove the tuberosities with the articular surface, so as to obviate all risk of future dislocations, and also to get rid of the insertions of the small muscles around the capsule; for, as you may be aware, when dislocation does take place, the supra-spinatus, as demonstrated by Sir Astley Cooper, offers the chief impediment to reduction.

Please notice the shape of the bone. It is quite unanatomical, a deep and wide notch presenting where the margin of the head had been in the habit of resting against the border of the glenoid cavity.

My late clinical clerk, Mr. Ashdown, who has very carefully recorded this case, says that at the time of the operation I was so surprised at the great gap at the head of the humerus that I searched in the capsule for a detached piece. He is quite correct: I thought that a piece must have been chipped out of the bone in one of the patient's falls.

Let me pass round for your inspection some woodcuts in this, the twelfth volume of the "Transactions of the Pathological Society." They illustrate a paper by Professor Flower upon the pathological changes produced in the shoulder joint by traumatic dislocation. You will notice in fig. 20 that the head of the humerus from an unreduced dislocation is deeply grooved by pressure against a non-articular part of the scapula, very much as is the head of the bone which I removed from the epileptic man. But, as I have already remarked, the strange point is that so great a change should have been wrought upon the head of the humerus by such a comparatively short-standing dislocation as that of my patient.

Mr. Southam's interesting case, which first directed my attention to the advisability of performing resection for frequently recurring dislocation of the humerus, was briefly this*:—A woman, æt. 45, had for ten years been liable to epileptic fits, and in one of them she had dislocated her left humerus. She had since dislocated this humerus upwards of fifty times—always beneath the coracoid process—and usually during the fits. She had worn a leather shoulder cap, but with no good result. On examination under anæsthesia the luxation could easily be produced and reduced (so also was it in my case). At the

operation the humerus was found healthy, but the anterior part of the glenoid cavity was deficient. Mr. Southam sawed through the humerus at the anatomical neck. (In my case the bone was divided just below the tuberosities). At the end of a year there had been no recurrence of the dislocation, and the joint was serviceable for all her ordinary household duties. In a note which I received the other day, Mr. Southam informed me that the case had continued to be perfectly satisfactory. Let me specially call your attention to the fact that the anterior part of the glenoid cavity was deficient in this case.

A valuable paper was read by Sir William MacCormac, at the French Surgical Congress, last April, upon the treatment of old luxations of the shoulder. In the case which he reports he cut down upon the bone and replaced it in the socket. He says, "Sir Joseph Lister having assisted at the operation, it is useless to add that all the anti-septic precautions were observed." Nevertheless, profuse suppuration and rise of temperature followed, and he ended by excising the humeral head through the *anatomical* neck.

In recording another case, in which he had resected the humerus for an old-standing luxation, Sir William MacCormac remarked that he found that the anterior part of the glenoid cavity had disappeared under the influence of the pressure exerted by the head of the humerus, and that the head of the humerus was there marked by a deep groove devoid of cartilage. The dislocation had existed for eighteen months.

It is interesting to note the effect of mutual pressure upon surfaces of bone which have not been differentiated for that purpose—which have not been protected and strengthened by a thin but resisting layer of articular bone and cartilage. In Mr. Southam's case the anterior part of the glenoid cavity had disappeared under the influence of unwonted pressure. In my case, as I show you again, the unusual pressure had altered the form of the head of the humerus very much as if it had been clay in the hands of the potter. In Sir William MacCormac's case, both the glenoid cavity and the head of the humerus were misshapen. It is strange that in Mr. Southam's case and in mine, there should have been so much change of shape, for, according to the histories, the head of the humerus lay in its abnormal position only occasionally, and then not for long at a time.

* "Brit. Med. Jour.," June 4, 1892.

Resection for fracture with dislocation.

The last case, which I have to bring before you, is that of a lady's maid, 40 years of age, whom I now call in. Six weeks before her admission she tripped whilst walking, and, putting out her left hand to save herself, damaged her arm and severely hurt her shoulder. Her medical man in the country examined her on two occasions under an anæsthetic, but he could not assure himself as to the exact nature of the injury. Ultimately, he put the arm up in a splint, and fixed it to the side. On her admission into the hospital, the arm was quite stiff and very painful; there was much thickening about the head of the bone, which could be felt in the arm-pit. There was anæsthesia of the left hand. The wrist and fingers were absolutely stiff, and there was about a third of an inch of shortening.



Fracture through anatomical neck.

The diagnosis being obscure, it was agreed that an exploratory incision should be made, and that, whatever else were done, the brachial plexus should be relieved of pressure. An incision was made through the anterior fibres of the deltoid, and the articular surface and the head of the bone were found in the subcoracoid region. There was a complete fracture through the anatomical neck with some comminution of the tuberosities. The humerus was cleared of the small muscles and sawn just below the tuberosities. The wound promptly healed, and, with kind assistance of Mrs. East, who subsequently treated her by massage and manipulations, she has recovered not a little of the use of the arm. She can, as you see, get her hand up to the back of her head, and she is able to use her fingers, which, previously, were stiff and unpromising. I am more than pleased with the way in which the case has turned out,

and, what is of perhaps greater importance, the patient herself is not only satisfied but grateful.

The practical points which I specially wish you to remember in connection with these clinical remarks are as follows:—Obscure injuries to the shoulder should be examined under an anæsthetic, and without delay. It is easy to overlook a dislocation of the humerus, and especially so in the case of a stout patient.

Old-standing dislocations should be treated with great discretion, as violence may entail a fatal result. A clean incision on to the head of the bone is not a serious operation, and either with or without resection of the end of the bone, it may give a very serviceable joint.

As regards what may be called an "old-standing" dislocation, I would suggest "anything over eight weeks."

TWO LECTURES

ON A CASE OF

UNILATERAL BRONCHIECTASIS,

With Remarks on the Pathology, Diagnosis, and Treatment of Bronchial Dilatation.

Delivered at St. George's Hospital by

WILLIAM EWART, M.D. Cantab., F.R.C.P.,

Physician to the Hospital.

II.

PATHOLOGY AND TREATMENT.

THE following remarks will deal with bronchiectasis in general, but special reference will be made to the unilateral form, and a parallel will be drawn between this and Corrigan's cirrhosis.

ÆTIOLOGY.

Predisposition and heredity. Can a liability to the disease be inherited? This is a question for future study; but we may conclude from the absence of any definite statement in most of our authors, that facts do not point very strongly to this influence. *Definite proof is also wanting as to predisposition*; but there is much in favour of its being present. We have in support of this view the analogy of the natural predisposition of some constitutions to affections of the arteries, veins, or lymphatics, and, in emphysema, of the alveolar

tissue itself. It may well be, therefore, that the peculiar aspect of sufferers from bronchiectasis is, in part, the expression of their predisposition, although chiefly a product of their disease. However this may be, we have reason to doubt whether any predisposition would mean more than a lessened resistance to conditions which are successfully resisted by sounder subjects.

THE MECHANISM OF PRODUCTION.

Almost invariably, whenever organic tubes dilate,

of a syphilitic ulcer, a large bronchus becomes narrowed, bronchiectasis is very apt to be developed. In this case there is an obstruction ahead. Of the two alternate aërial currents, it is the expiratory which is obstructed—the condition represented in the diagram. Forcible expiration would set up a relatively high pressure within all the bronchi below the stricture, whilst, during inspiration, the pressure would be rather less than normal, owing to the delayed supply of air. (The relative strength of the inspiratory and of the

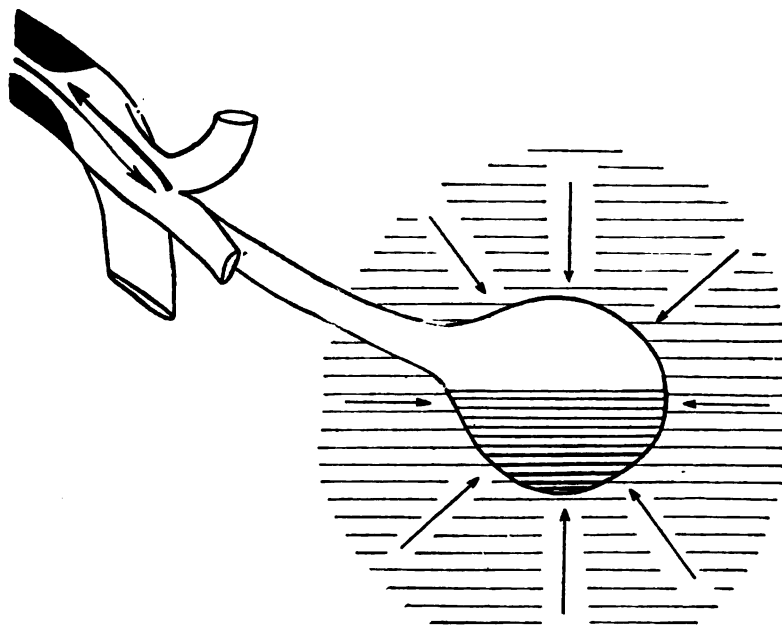


FIG. 3. Diagram of Bronchiectasis due to Stenosis of the Bronchus.

The stenosed left main bronchus is seen dividing into the bronchus for the lower lobe, and a large bronchus for the portions of the left upper lobe corresponding respectively to the right upper lobe, and to the right middle lobe. The bronchiectasis involves a branch from the latter division.

The thick arrow indicates the alleged intra-bronchial pressure arising during cough. The converging arrows represent the direction of the expiratory forces.

the cause is *obstruction*. We must look in bronchiectasis for obstruction somewhere; and we shall seldom fail to find it. The bronchial tract differs from other animal tubes in that the current within it is not steadily moving in one direction, but alternating. Obstruction may thus be set up within a bronchus in more than one way: and in relation to any given dilatation the obstruction may be situated either *in tergo* or *in fronte*. This I shall hope to explain to you.

I. BRONCHIAL STENOSIS. *Obstruction in fronte*. When, as sometimes occurs from the cicatrization

expiratory pressure is indicated by the thickness of the arrows.)

There is another factor besides the respiratory oscillation of pressure. Occasionally, in the lung below the stenosis, you may find a thin-walled sacculation, completely filled like a cyst by stiff gelatinous mucus. Air in this case has become entirely displaced by secretion, and it is absolutely excluded from the corresponding pulmonary district. The obvious inference would be that bronchiectasis had arisen in that instance from accumulation of secretion within the tube; and this is rendered

the more probable, as the swiftness of the expiratory tracheal current, which we all know is essential to an effectual cough, is much lessened by the existence of stenosis, and the lung therefore is less able to clear itself of mucus. In this way arrears may grow, and permanent distension may result.

II. BRONCHIAL CATARRH. *Obstruction a tergo*;—*The inspiratory and expiratory theories.* Although the derivation of bronchiectasis from bronchial catarrh is undeniable, the mechanism at work is not manifest, and the explanations given are multiple. This is due to the complexity of the tissues and of the forces at work. It is difficult to

either of the two other coats, (2) *the muscular*, and (3) *the fibrous*. He argues from the infrequency of bronchiectasis, that something more is needed for its production than long-continued and violent cough, and than powerful intra-bronchial pressures, to which all of us are constantly exposed. He regards the affection as mainly due to a process of peri-bronchial fibrosis, invading the fibrous and the muscular coat.

Local failure of muscular tone or local muscular spasm should also be borne in mind as possible agents, especially in the spasmodic affections, asthma and pertussis.

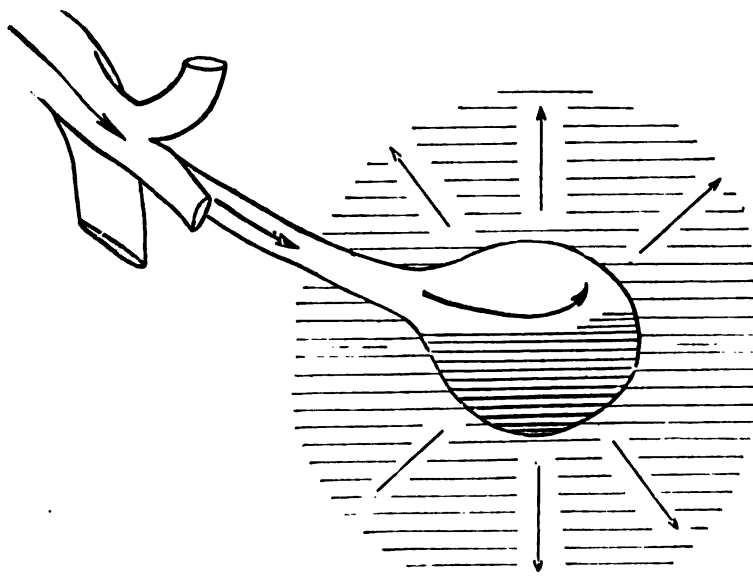


FIG. 4. Diagram illustrating the "inspiratory" theory of Bronchiectasis.

The diverging arrows represent the direction of the inspiratory forces.

The shaded zone surrounding the bronchiectasis is supposed to be more or less deprived of air.

fix upon any one of them the original fault without which the pulmonary machine would continue to work true. The flaw may reside in the substance of a bronchus, or in its relations with other parts; that is, the origin of bronchiectasis may either be (a) structural or (b) mechanical.

(a) *The structural or mural origin of bronchiectasis* has been insisted on by Lebert.* Whilst admitting with all other observers some degree of local affection of (1) *the mucous membrane*, as a result of bronchitis, Lebert points out that inflammation and interstitial changes may involve also

* *Loc. cit.*

(b) *The mechanical or respiratory theories* are the inspiratory and the expiratory. As in the case of emphysema, their discussion has hitherto been carried on in a somewhat inconclusive manner. The question is, How do bronchitis and bronchial catarrh affect the dilating bronchus otherwise than by local inflammation: this question is variously answered.

(1) *The inspiratory theory* may be identified with Laennec. He regarded bronchitis as the chief cause of bronchiectasis in connection with the accumulation of secretion, which necessitates increased inspiratory efforts to clear it away. This

theory affords a workable explanation, so long as it is understood that atmospheric pressure has lost access to the alveoli *beyond* the dilatation, and that the same pressure is no longer bearing on both faces of the bronchial wall. This condition would be brought about by any obstruction within the small bronchi which are situated beyond the sacculations. The diagram (fig. 4) illustrates this theory, without thoroughly explaining it, just as the other diagram (fig. 5) fails to prove the correctness of the expiratory theory.

(2) *The expiratory theory.* The possibility of cough dilating a bronchus has often been argued. You are aware, however, that although the intra-

lobules would upset the even balance between the intra- and the extra-bronchial pressure, and in delicate and predisposed subjects, the bronchial wall might yield and become permanently dilated. *Pulmonary atelectasis* is thus, in relation to these theories, as it were a working screw, without which they fail to explain the mechanical result.

THE RELATION BETWEEN EMPHYSEMA AND BRONCHITIS.

Atelectasis without thoracic complications (deformity or abnormal contents) always means compensatory emphysema. Thus the cause which produces bronchiectasis also gives rise to some

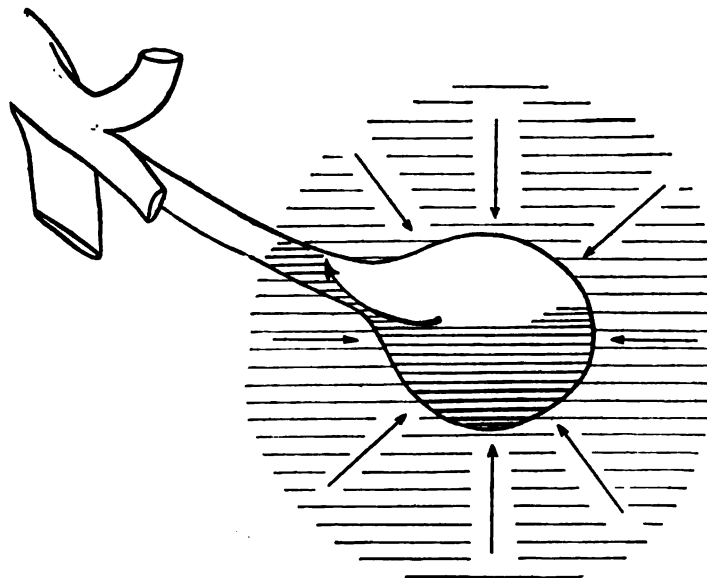


FIG. 5. Diagram illustrating the "expiratory" theory of Bronchiectasis.

During cough some of the viscid accumulation may set up temporary obstruction as represented.

bronchial pressures are occasionally great in healthy subjects, no harm ensues when forcible expiration is practised with closed glottis. Nothing gives way within the visceral cavities under the high pressures due to muscular strain, although their *outer* wall may yield, as in hernia. The reason is that pressure opposes pressure; and that often very delicate structures—a striking instance of which is the fringe of the semilunar valves—are protected by the evenness of the high pressures bearing equally on both their faces, whilst a fraction of the same pressure would suffice to rupture them if applied to one side only. Now, in the case of any bronchus, airlessness of an adjoining lobule or

emphysema in a secondary fashion. But you will notice that there is, as it were, a complementary relation between the amount in which the two changes develop. It is remarkable that in whooping-cough and in asthma—both noted for their viscid secretion—many suffer from permanent emphysema, but comparatively few develop bronchiectasis. The reason for this lies near. If emphysema can be developed on a sufficiently large scale, the bronchi are protected from excessive inspiratory strain. If, on the contrary, through adhesions or atelectasis, the pulmonary tissue should expand imperfectly, the bronchi must dilate.

This principle serves to explain the specimens

which I have placed before you (see figs. 1 and 2, pages 263, 264). It also makes us understand how bronchiectasis, beginning in one part of the lung, may lead, especially in the predisposed, to its universal extension. Loss of adjustment between intra-alveolar and intra-bronchial pressures having led to bronchial dilatation at one spot, the finer adjustment of neighbouring parts will almost invariably suffer also.

THE ANATOMICAL VARIETIES.

Two chief varieties of dilatation are described—the *saccular* and the *cylindrical*. There is also a third, less important, the *bead-like* form.

If the collapse which we have described be *terminal*, then the *saccular* form will probably result; if the collapse due to bronchitis be *lateral* to the tube, then the *cylindrical* or the *beaded* form is apt to develop. But in the more progressive cases the disease passes readily from the one to the other type, and there is no strict distinction between the two sets of changes. The terminal, globular, thin-walled pouches are more often seen in the midst of spongy tissue, and belong to the earlier stages; the cylindrical dilatations, approaching the pulmonary root more closely, are often surrounded with fibrous tissue; they are seldom absent in the later stages. If the tissue surrounding the early lesion should take on emphysema, the condition may remain stationary as regards the size of the dilatations; but if broncho-pneumonia with lymphatic irritation and interstitial fibrosis should be set up, the entire lung, or, as in the second specimen placed before you, the part affected may gradually undergo complete fibrosis. We may conclude that in any individual case the more fetid the sputum, so much the greater will be the fibrous change, especially in the late stages; and the greater the fibrous change, so much the greater the pulmonary atrophy.

In the later stages the mucous membrane commonly loses its sensitiveness, and large accumulations occur. It is conceivable that insensitiveness of some portion of the bronchial system may occasionally occur early, and, indeed, may have some share in the causation of the disease. Concerning this point, further information is needed. An agency of this kind would fit in with the theory, already mentioned in connection with stenosis, which refers the origin of bronchiectasis to the weight of delayed secretion, rather than to an uneven distribution of atmospheric pressures.

PARALLEL BETWEEN UNILATERAL BRONCHIECTASIS AND CORRIGAN'S CIRRHOSIS.

We can now briefly contrast these two affections under their various aspects.

ÆTIOLOGICAL AND PATHOLOGICAL DIFFERENCES.

The *determining cause* of Corrigan's cirrhosis is probably pleurisy in most cases; that of bronchiectasis is more often bronchitis and catarrh. In bronchiectasis the *dilatation* is the earliest change; in Corrigan's cirrhosis it is altogether secondary to other changes. The *fibrosis* is the essential and earlier change in cirrhosis: in bronchiectasis the first tendency is to collateral emphysema of the unaffected parts; fibrosis follows only when much irritation is set up by overflow from the dilated sacs into more or less distant bronchial divisions and districts.

CLINICAL DIFFERENCES.—(1) *In aspect* the two sets of cases are very different, the cirrhotic patient being usually pale and dry, the bronchiectatic, as previously described, rather inclined to venous fullness.

(2) *The thorax*:—The strong adhesions which exist in Corrigan's cirrhosis preclude the expansion of the sound lung into the other side of the chest; the thorax becomes much deformed, and its movements limited to one side. In unilateral bronchiectasis adhesions are usually present, pleurisy having occurred in most cases; but they are less universal and not so dense. If adhesions should be absent from the upper part of the chest, as in the specimen before you, this would allow considerable encroachment of the sound lung into the opposite side; and loss of symmetry of the thorax would be to a large extent prevented. This result is not uncommonly observed in unilateral bronchiectasis. An additional reason for the slightness of the thoracic deformity lies in the very gradual progress of the pulmonary changes, emphysema or hypertrophy being able to keep pace, up to a certain point, with the pulmonary degeneration.

DIFFERENCES IN PHYSICAL SIGNS. Time would not permit me to enter into the full detail of this part of the subject, of which a sketch has already been put before you in my first lecture. Let me only point to the great difference observed, between typical cases of the two affections, in relation to the intra-pulmonary sounds. In bronchiectasis the respiratory murmur is seldom entirely absent; in cirrhosis it is completely abolished. Again, the cavernous sounds in bronchiectasis are extremely

moist and active; in cirrhosis they are apt to be of a much drier type. These distinctions will be of practical help in diagnosis; but the latter will need to be supported by a careful study of the other physical signs.

PROGNOSIS.

The disease is incurable in its advanced forms; but it is still a question whether the earliest stages may not be recovered from. By some observers whooping-cough is held to sometimes give rise to a temporary bronchiectasis: this would be a minor degree of the affection, and recovery would be favoured by the elasticity, by the growth and by the nutrition which belong to childhood. Although incurable, bronchiectasis is not usually fatal in itself. Its complications alone are fatal. Of these the most frequent is septic infection following upon decomposition of the bronchial contents, and in some cases ulceration of the cavities. The septic conditions may develop in various directions, pneumonia and intracranial abscess being among the most frequent terminations.

The affection may last for years without great impairment of health, except as to respiratory efficiency. The tendency is towards progressive implication of the lung; but the rate of this progression is a very variable one, and is governed by many circumstances, the detail of which we cannot now enter upon. What has been said concerning the mutual relation between emphysema and bronchiectasis clearly points to the value of pulmonary hypertrophy in checking the progress of bronchial dilatation.

From *phthisis* bronchiectatic patients have least to fear. I shall not refer to the local and secondary bronchiectases, nor to the often spurious form of dilatation, incidental to excavating disease of the lung. Cases of pure bronchiectasis are protected from the danger of pulmonary tuberculosis equally by the fibrosis and by the emphysema, which are secondary to the affection.

Cancer, according to Barth,* occurs in a high percentage among sufferers from bronchiectasis. This is a point deserving future investigation; enough for me to remind you that the patient who has been the subject of this lecture, died of abdominal cancer.

TREATMENT.

We should not be discouraged by the incurable character of the affection from endeavouring to

mitigate its evils, and, if possible, to stay its progress. *Principiis obsta*;—in no other disease is it more important to treat early.

The chief indications in bronchiectasis are:

- I. To empty the cavity.
- II. To check the secretion, that is, to lessen the catarrh.
- III. To bring about, if possible, contraction of the dilated tubes.

I. Mechanical means should be adopted to promote evacuation of the pent-up secretion. This result is sometimes favoured by posture. The patient will learn in which position he is best able to cough up the secretion. Gerhardt suggests systematic compression of the patient's chest during expiration; and this measure may be recommended.

II. With a view to checking the catarrh, treatment must follow various directions.

(1) *The climatic indication* is, of all, the most important. Patients should be made to reside under climatic conditions which will reduce their catarrh to a minimum; and every adverse influence capable of inducing fresh attacks should be carefully avoided. *Evenness* of temperature is of more importance than mere *warmth*; and *dryness* is essential, unless the moisture of the air be *salted*, as in sea-air, or in the saline atmospheres provided at certain health-stations inland.

(2) The judicious use of *sulphurous waters* may be combined with the climatic indication at such stations as Eaux Bonnes, Cauterets, Aix les Bains, etc., or, failing this, the occasional administration of Sulphur may be followed by beneficial results, due to the well-known property of Sulphur of checking excessive suppuration.

(3) *Medication* will effect much; but it will be necessary to vary the agents employed. The most effectual medicines are those belonging to the turpentine group, and to the balsams. Their stimulating effect is due to the active principles being in part exhaled by the bronchial mucous membrane. Turpentine in its various forms, or Eucalyptol, Carbolic Acid, Tar, Creasote, etc., may be given in emulsion or some other form; but frequent changes are needed in order to keep up the effect.

(4) Most of the drugs which have been enumerated may be suitably administered by *inhalation*.

(5) *Tonics* are required to combat any tendency to anæmia; and a strengthening diet is rendered necessary by the constant drain from the lung.

* "Dilatation des Bronches," Paris, 1856.

III. Having secured the first two points, our treatment should address itself to *reducing*, if possible, the size of the *dilated tubes*. The only agency which could take real effect in this direction would be an improved ventilation of the lung, which would favour the true hypertrophy, or at any rate the enlargement of the sound parts of the lung. This is an aspect of treatment which is, perhaps, too much neglected. For the attainment of this increased respiratory function the means at our disposal are of two kinds:—

(1) *Passive* exercise of the chest. This is a valuable aspect of the mechanical treatment which Gerhardt recommended for the relief of the catarrh. The object in this case is different, and the method will differ from that mentioned above. The chest must be exercised not only during expiration but also during inspiration, according to the usual method.

(2) Of yet greater value are the *active* pulmonary gymnastics which are always associated with open air pursuits. Granting that the patient has been placed under favourable climatic conditions, an open air life is strongly to be recommended; and gradually increasing physical exertion may be enforced with the promise of as much success in suitable cases, that is, in those still possessing the advantage of youth, as is compatible with the profound structural alterations which have taken place.

A CLINICAL LECTURE

ON

SUPPURATION IN THE SULCUS LATERALIS.

Delivered at University College Hospital, Jan. 9, 1894, by

ARTHUR E. BARKER, F.R.C.S.,

Professor of the Principles and Practice of Surgery at University College, and Surgeon to the Hospital.

GENTLEMEN,—Before giving you the history of the case of this boy who was admitted here on December 28th, I should like you to see—so far as you can, since he is now convalescent,—what I have done. Behind the ear you will observe a semilunar incision, beginning behind the pinna high up, sweeping downwards and backwards, and ending well below the mastoid process behind.

This incision is now nearly healed; we have had it drained at the upper and lower angles, and in the middle the drain lies over an opening in the bone. The incision is the usual one,—from the upper part of the pinna right round in a curve. In making it you cut across the posterior auricular vessels transversely, but they are easy to stop. You turn down the flap, and expose the whole mastoid region. This is the incision I always make now; I have tried many others.

This is a case of suppuration in the sulcus lateralis, that groove on the inner aspect of the mastoid portion of the temporal bone which lodges the lateral sinus. This, for several reasons, is a very grave condition. First, it is an intracranial suppuration for which there is no natural means of exit; and pent-up suppuration in any place is grave, but in this situation it is more than usually so. Here it may give rise to mischief in a great variety of ways that will at once occur to you. The inflammatory focus may spread in area and in depth, and may then affect deeper structures. Thus it may affect the meninges. Passing through the dura mater it may cause *chronic* pachymeningitis or *chronic* leptomeningitis; or it may set up acute pachymeningitis or acute leptomeningitis. The inflammation may be localized or diffuse. It very often gives rise to intracranial abscess; abscess, that is, either in the cerebellum or in the temporo-sphenoidal lobe; and this constitutes another risk. Then it may produce septic phlebitis of the lateral sinus, which also may be dangerous for several reasons. The organisms which are producing the septic change outside the vein may gain access to its interior and charge the whole blood stream with septic material, producing septicæmia. Or from this inflamed base clots containing organisms may be dislodged, and becoming impacted in the capillaries of the lungs there start a septic pneumonia, as I have seen them do. Again, this septic phlebitis may be dangerous by producing thrombosis of the lateral sinus; and this may be serious not only from the blocking of the sinus, but from its extension downwards into the deep jugular vein, and so into the lung. Those are the dangers to be borne in mind as liable to occur,—meningitis, cerebral or cerebellar abscess, phlebitis of the lateral sinus, and septicæmia or pyæmia.

A word as to the causation of this condition. In the vast majority of cases it starts from disease of the middle ear. Disease of the middle ear, as

we all know, is exceedingly common. If you examine any analysis of a large number of deaths (e.g., the Registrar-General's returns) you will find that those from disease of the middle ear and its complications are very numerous. As to the kind of disease of the middle ear which gives rise to this phlebitis of the lateral sinus or inflammation in the sulcus lateralis,—if you take all the known diseases of the auditory apparatus, including those of the internal, middle, and external ear, you will find (I am drawing now upon a very large mass of statistics which I accumulated some years ago) that diseases of the external ear make up about 25 per cent. of all; that diseases of the internal ear constitute about 8 per cent., or thereabouts; while diseases of the middle ear account for 67 per cent. Now, it is amongst these diseases of the middle ear that we look for the source of this affection. But when you come to analyze this 67 per cent. of all known diseases of the middle ear you will find that about 30 to 35 per cent. are suppurative, the other half being made up of chronic dry catarrhs, etc., of the tympanum and Eustachian tube.

Amongst these suppurative diseases of the middle ear you have to distinguish between two great classes—the acute and the chronic; but I may tell you (and this is about the most important point I can draw your attention to in this connection) that this condition rarely, if ever, starts from *acute* middle ear disease. That is commonly forgotten, and is the source of a great deal of mischief in treatment. It always starts from chronic disease of the middle ear. Now, of all suppurative diseases of the middle ear, by far the largest proportion of this 30 per cent. or so, namely, 25 per cent., are chronic conditions as we see them, while about 5 per cent. are found to be acute. It is amongst these chronic conditions, then, that we seek the lesions which give rise to this intracranial suppuration. Now, why is that? The reason is probably this:—In acute cases (I am talking of first attacks, of course) the middle ear is still lined by a mucous membrane which is furnished with its own circulatory system and its own lymphatics. Septic matter having been formed on the surface or in the tissue of that membrane is probably carried off and disposed of by the lymphatics. That the lymphatics can carry off a great amount of septic matter and to a certain extent guard the deeper structures from septic infection we know, because we often find the neighbouring glands much enlarged, and they often suppurate after

acute attacks of middle ear catarrh. As long, then, as the mucous membrane of the middle ear is intact, or more or less intact, there is very little danger of the disease spreading to any intracranial structure whatever. But, when this has been destroyed, and the surface of the bone, denuded of its mucous membrane and periosteum, has been exposed and inflamed, then it is that the mischief is likely to start. That is a most valuable point to get clearly into our minds, because, knowing that, we shall always be careful to inquire into the history of these cases, and to ascertain whether the disease is a *primary* acute or a *chronic* attack. Of course, acute attacks may be primary and acute, or they may be secondary or tertiary acute. That is to say, you may have an old mischief in the ear which has destroyed all its mucous lining, quieting down and remaining for years without giving any trouble, at the end of which an acute inflammation may be grafted on it. That, of course, may become dangerous, and produce an intracranial complication; but I am talking now of the distinction between primary acute attacks where the mucous membrane is still intact, and those cases where it has gone. You must inquire into all these cases, and find out whether the patient has had anything of the kind before in his ear; whether he has had previous attacks like this; or whether this last attack is simply a lighting up of some old mischief. If it is the first, and a primary acute onset, you need not anticipate an intracranial inflammation, as a rule; if it is an attack which has become chronic, or a chronic case which has been reinfected in one way or another so that a fresh acute process has been started, you may admit the possibility of this particular mischief, as in the case before us.

How does the inflammation spread from the middle ear to the sulcus lateralis? It is very important to understand this. Owing to the chronicity of the condition the mucous membrane has been destroyed, and the bone exposed; so that the chronic inflammation in the middle ear produces a chronic osteitis all round the tympanic cavity. Now, the vessels of the tympanic cavity have, under normal conditions, a tolerably free anastomosis through the thin bone with the vessels of the dura mater, and these vessels of the dura mater, of course, communicate with the sinuses. What happens is that the septic disease in the middle ear spreads along the smaller radicals of the veins which pass between the

middle ear and the dura mater, the disease extending in the form of a septic phlebitis; or it may spread directly by caries and destruction of the bone. The process commencing in the middle ear passes, unchecked by any special lining, steadily through the bone until it reaches the dura mater, when it forms a collection between the latter and the bone. Then, of course, the same inflammation may spread upwards under the dura mater of the petrous portion of the temporal bone, or backwards towards the posterior aspect of the latter, which is the part we are considering now, the sulcus lateralis.

The history of the onset varies a good deal. Sometimes the account given is that months or years previously the patient had an acute attack in the middle ear which had gone on discharging and become chronic; that the ear has become blocked up by polypi; but that very little trouble has been experienced until the patient has caught a cold. An acute pain is thereupon felt, as was the case with this patient, in the middle ear; the patient's temperature rises, and he feels very ill; he probably has a chill, and then the symptoms which we shall describe presently set in. On the other hand, there may be no evidence whatever of blocking in this old chronic mischief in the ear, which may have lasted for years. In this particular case the boy had the original attack from which his present condition ultimately arose, seven years ago; but up to the onset of his present illness he has had no special mischief in his ear,—no polypi and no blockage. When we look into the meatus, in this case, we find no trace of either membrane or ossicles; there has been nothing to prevent the discharge from flowing away, such as it is; but the boy gets a so-called chill from a cold blast of air, and suddenly acute symptoms of inflammation start which rapidly spread inwards. That I have always thought a clear instance of direct infection,—such an infection as we get in erysipelas. There is no blockage; there is no discharge pent up; but there is inoculation on the raw surface with some septic cocci, which find a perfectly suitable soil for their growth there, and so, developing with great luxuriance, they spread along the inflamed vessels through the bone into the sulcus lateralis. That is, perhaps, the commonest course of such cases.

As to the symptoms, what we commonly hear of in cases of mischief in the sulcus lateralis after such a history as that is that the patient has had

a rigor to begin with, after which the temperature goes up as high, it may be, as 103 or 104, and, as in a private case I saw some time ago, even as high as 105, or nearly 106. The temperature usually remains up for some time, and then oscillates. The patient has a flushed face and rapid pulse; he feels very ill, and sooner or later he begins to feel a little drowsy or heavy. On examining the ear you may find no drum, and no obstruction; or you may find polypi blocking up the meatus. On inspecting the mastoid region in a well-marked case you will usually find a good deal of swelling, evidently very deep seated, affecting the periosteum of the bone; there is also a good deal of tenderness. On pressure you find the swelling to be due to oedema of the part. That was present in this case. There was a diffused hardness over the mastoid process of an oedematous character, extending not very far below the mastoid process, but chiefly over it. If you tap the part you will find that it is very sensitive.

In all this, however, we have only the symptoms of retained pus. There are really no distinctive signs beyond those I have given you which will help you. In an uncomplicated case, a case without meningitis, cerebral or cerebellar abscess, or phlebitis of the lateral sinus, the symptoms are simply those of retained pus somewhere in that neighbourhood. Of course, if other complications exist, there will be certain fairly indicative symptoms to guide you; but in a simple case there will be none such. By what, then, are you to be guided? You will be so chiefly by your previous knowledge of what these attacks of middle ear catarrh are likely to develop into, and by what you find in the middle ear. First of all, you are helped by the question as to whether it is acute or chronic. If it is chronic you are to a large extent guided by the appearance of the ear. If there is blockage, with granulation or polypi and pent-up discharge; if that has been for a long while present; if there is continued high temperature, even after removal of the polypi and washing out of the ear, you begin to suspect some deeper-seated mischief. If, on the other hand, you find the membrane and ossicles gone, you may see if the condition of the middle ear is one of violent inflammation; you may see the character of the secretion,—a thin ichorous pus, of the kind usual from septic foci; and as there is no pent-up matter there you begin to think that it must be pent-up somewhere deeper,

and the most likely place, supposing you can exclude the glands of the region, is the mastoid cells or the sulcus lateralis.

But supposing that there are any other complications, then you have certain signs. I cannot go into all these particularly now; but you will look out for them, such as they are—and they are not very distinct—of cerebral abscess and meningitis. If you can exclude these then you are helped largely. Then you look out for the signs of septic phlebitis and thrombosis. If there is septic phlebitis the rigors will usually persist; in fact, with septic phlebitis there is almost typical pyæmia or septicæmia,—rigor after rigor, with an oscillating temperature. If there has been only an initial rigor, with an oscillating temperature afterwards (and remember you need not be in a hurry about these cases), you are pretty sure there is no marked phlebitis of the lateral sinus, anyway. If there has been a mild attack of phlebitis of the lateral sinus there is the same temperature, and almost certainly there will be thrombosis of the sinus, because an inflamed vein almost invariably collects upon it the fibrin of the blood; and certain symptoms will thereafter ensue. In typical cases the thrombosis is pretty sure to extend to within reach of your finger. A hardness can generally be felt over the deep jugular vein, and distinct tenderness where the vein is blocked by the thrombus. In addition, the side of the face may be flushed to a greater extent than the other. Close examination of the eye on the affected side will reveal an engorgement of the vessels of the sclerotic, from a retardation of the return of blood from the deeper parts of the eye. These are the symptoms upon which you would rely for help in the diagnosis of thrombosis in the lateral sinus.

As to the other special symptoms,—you will sometimes get optic neuritis. I have seen a case in which this was present, and which we had reason to believe was nothing more nor less than one of suppuration in the sulcus lateralis. When I let out the pus the optic neuritis, which was most intense, subsided, and the patient got perfectly well. There is, very often, at the same time some sickness, but that is the sickness which accompanies all septic infection of the system; and there is nothing special about it here.

There is not, then, a great deal to guide us; there is nothing very characteristic about these symptoms. I have given you those that are, to my mind, most characteristic; but they are not

very much so. You will learn more from a study of these affections as a whole, and especially if familiar with the appearances to be found in the middle ear from the conditions there, than from any special symptoms; you will learn what to expect, and what the case is likely to develop into.

If there is still doubt, other measures have to be taken to clear it up; and where the patient is so seriously ill as these patients usually are, with a high temperature and commencing drowsiness, and with the prospect of the disease developing into something serious, you are justified in going to some considerable length. The best plan if you suspect pent-up matter is to search for it. First of all, you have, as a matter of course, thoroughly cleared out the middle ear. In some cases you may have time to wait; if you can wait for a day or two it is well to devote that time to the thorough cleansing of the ear, removing granulations, drying it, dusting it with Iodoform and Boracic powder, and so getting the part into as cleanly a condition as possible. In some cases, if the disease has only just begun, if you effect free drainage from the middle ear, and get the part thoroughly clean, the temperature will steadily come down, and the symptoms of septic absorption disappear. In this way you may avert suppuration under the dura mater at the sulcus lateralis. If there is time, leeches may also be applied over the mastoid process; and in that way also the disease may be cut short. At the same time, also, internal remedies are used,—Quinine, Iron; and purgatives. That is, if you have time. But the patient may have rigors; the pain and swelling may be increasing; and he is getting drowsy. You must then go further and search for the pus in the most likely situations. Now it is quite possible, if the patient be an adult, for all these symptoms of retained pus to be caused by its retention in the mastoid cells. In the child, where there is only a mastoid antrum and no cells other than rudimentary ones, you do not expect pus in the mastoid process. But in an adult there may be pus in this situation, and, as this may be the source of all the mischief, you at once expose the mastoid by the incision I have shown you. You make for the place where the matter is most likely to be pent-up. That will be somewhere about the termination of the mastoid antrum. The mastoid antrum is an offshoot of the middle ear; its floor is about level with the roof of the meatus, its roof with the roof of the

tympa-num. In a child, such as our patient, it is a little cavity about half an inch long and a quarter of an inch broad, which would about hold the end of a slate pencil, and which passes directly backwards. In the search for pus you make for that in the first instance. To get at it, the best line of incision, through the soft parts, I can give you, is a curved one, commencing just above the pinna, and running backwards and downwards. You turn down the flap, and then the question arises where best to chisel or trephine the bone. In operating, I select the following measurements. From the middle of the meatus I draw a horizontal line backwards; just at the insertion of the pinna behind I draw another vertical line at right angles to that; and in the angle which these form, that is, above the horizontal line and behind the vertical, I begin to chisel the bone. This lands me in the mastoid antrum. It comes to this, that it is about half an inch above and half an inch behind the centre of the meatus. If you chisel there you will almost certainly come upon this cavity. You soon get into the antrum, especially in the child, where the covering is thin. You make out whether there is any pus. If there is a large collection it may be washed out through the external ear. But in some cases you find nothing that would account for the symptoms; then, if the patient is an adult, you seek for the mastoid cells; that is to say, you begin to chisel away the bone downwards and backwards. In some adults these are very ill developed; and you must be exceedingly cautious. If you chisel away and there are no mastoid cells, you come upon the sulcus lateralis; and, although you want to open that if necessary, you do not want to do so by an unwary cut, which may open the lateral sinus too. Having opened the mastoid cells and failed to find pus pent up there, you must proceed to chisel away the inner table of the mastoid process until you have opened up the lateral sulcus. That you can do with perfect safety in cases where the lateral sinus is not rotten. You can pick away the bone with the chisel and bone nippers until you have opened the sulcus. If there is pus it will flow out readily, as it did in this case. All you have to do is to secure a large enough opening to evacuate the secretion and the operation is over.

But if, as in an advanced case, after exposing and probing the lateral sinus, you find that it is thrombosed, it may be your duty to go further. You know that that thrombus is very likely sooner

or later to break up, and if it breaks up it is but a short course for it to reach the lungs, where, being septic, it may set up bad pneumonia. In a case like that the best plan is to expose a considerable portion of the lateral sinus, and ligature it above; then to cut down in the neck, and ligature the deep jugular vein; then, having got your ligature above and below the thrombus, you incise the sinus and turn out the clot, washing it out if necessary, and drain it. But in a case like this, all that is necessary is to chisel away the bone until the pus begins to well out of the sulcus lateralis, and then to enlarge the opening. In this case, when I had exposed the lateral sulcus I could feel with the probe that the latter was quite sound, and that there was no evidence of thrombus; moreover, there was no evidence of thrombus beyond it,—no tenderness along the course of the deep jugular vein, no injection of the veins of the sclerotic, and no unilateral flushing of the face. If there was any affection of the lateral sinus at all it was simply a certain amount of phlebitis, but to a very limited extent, and merely from proximity to the septic focus; and that, we know, would subside as soon as the pus was let out. I went no further. I washed the part out, and drained it; and the child has got rapidly well. In this case no time was left for the characteristic temperature to develop. On admission it was 101; but usually it rises very high,—somewhere about 103 or 104, and after letting out the pus there was complete subsidence. After that there is nothing left to be done but wash out the part, feed the patient with low diet, keep him quiet in bed, and dress antiseptically.

The moral of our study of these conditions is, in every case of suppuration in the middle ear, whether it is acute or chronic, to be exceedingly careful; but especially should chronic cases be most carefully attended to, and acute attacks grafted on these old chronic conditions are the most dangerous of all. On another point I want to lay great emphasis. If you have a case like this where you suspect you will have to do some operation on the mastoid antrum, the mastoid cells or the lateral sinus, do nothing to it until you have thoroughly cleansed out the middle ear. This may be full of the most fearfully septic material which will probably infect your wound. Do this cleansing of the middle ear with the utmost caution. If you have time, the best plan is to dry it up, mop it out with Perchloride of Iron, and dust it

with Iodoform for a day or two before attempting to remove the granulations. If when you first see the case, you begin to scrape the ear which is full of septic material (and, remember, a great variety of pathogenic organisms have been found here), you may cause an increased amount of that matter to flow into the blood stream through the inflamed vessels. I have seen a patient who came with almost normal temperature to have his ear looked after for some mischief in it, made terribly ill by such procedure adopted before asepsis had been secured; and patients with granulations in the ear who have been doing fairly well on having these granulations at once scraped without any antiseptic preliminaries have got septicæmia and died. Therefore in the first place be careful to dry out the ear, and get it quite clean. That has been my rule for a great many years. I have had considerable experience of ear troubles, having for ten or twelve years presided over the out-patient ear department; and during that time it was my invariable rule in dealing with these chronic cases, for hours or days before I did any operation, to get them as clean as was possible. In cases where an exception has been made to that I have seen disaster threaten, the most virulent conditions being induced by meddling with the ear when it was in a septic condition.

There is a tendency at the present day to attack all these cases of suppuration in the middle ear with high temperature at once by operation: there is an idea afloat that in all these cases, when in doubt, you should open the mastoid cells. That ought not to be the routine treatment. To be obliged to open the mastoid cells on the mere chance of finding pus there is a confession of weakness; you must have good solid grounds to go upon before you adopt that practice. But if it is not to be routine practice, you will not know how to avoid it unless you study these cases just as carefully as you would study chronic ulcers of the leg, or diseases of the rectum. It is quite possible for a man studying general surgery to include one of these in his range of study without making it the exclusive province of the specialist; and if we are to avoid the evils that grow out of specialism it is by studying these diseases specially.

In this lecture I have given you some facts which I accumulated years ago; and had there been time I should like to have given you a good many more bearing on this particular case, showing

you how similar cases died, and the complications that arose. I have made a good many tables, and pointed out the positions in which these various foci may be searched for, and dealt with many other points. These you will find in the Hunterian Lectures which I gave some years ago in the College of Surgeons, on "Intracranial Inflammation due to Suppurative Disease of the Temporal Bone." In them you will find this drawing of a section of the temporal bone, to which I wish here to draw special attention, as it illustrates the points to which I have alluded in connection with the operation.

THERAPEUTICAL NOTES.

Hypophosphites in Defective Nutrition of Bone and Nerve Tissues.—Dr. L. G. Pedigo draws renewed attention to this very important subject. He gives three types of cases where he has found Syrup of the Hypophosphite of Lime of most marked use:—

1. Delayed dentition in ricketty, backward children.
2. The low, inflammatory conditions of bone affections in scrofulous children, excited by slight injury, and often the precursors of pronounced tubercular Potts', or hip-joint disease. In this type of case he remarks on the rapidity with which pain in the part subsides as evidence of a decided beneficial influence of the drug.
3. Syphilitic affections of brain or cord, after Mercury and Iodide of Potassium have exerted their specific absorptive influence. Here the drug seems to open up, as it were, the compressed nerve tubules, and puts fresh life into them.

He states that improvement in these three classes usually sets in very quickly, but advises a prolonged course of treatment as often the benefit is slow in making its appearance. In the third group the Hypophosphite treatment must not be begun too early, not until the Iodide and Mercury have done all they can.—(*Amer. Med. Jour.*)

Erysipelas of Face.—Dr. Tison advises the painting of the affected part with a saturated ethereal solution of Camphor. This acts as an excellent sedative, relieving the pain and inflammatory condition very rapidly. The painting may be repeated as often as necessary.

(*La Sem. Med.*)

THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 7, 1894.

A CLINICAL LECTURE ON SOME POINTS IN THE TREAT- MENT OF TYPHOID FEVER.

Delivered at King's College Hospital

By JOHN CURNOW, M.D., F.R.O.P.,

Physician to the Hospital.

GENTLEMEN,—In going round the wards just now, you saw two cases of typhoid fever which we have had an opportunity of watching for some time past. It will not be out of place, therefore, if I ask you this morning to consider the treatment of this disease as has been practised by me in now some hundreds of cases here and at the Seamen's Hospital, where it is almost always under treatment.

In the first place, I would ask your attention to severe pyrexia, meaning by this a continuance of a temperature of 103° or over for several hours in succession. When the temperature has once reached 103° , I instruct my house-physician to take it every half-hour for four hours unless it has fallen. If it rises to 104° and persists over 104° for three or four hours, he is to regard the case as a hyper-pyretic one, and treat it accordingly, for then a great and special danger arises on account of the heat-changes which then take place in the heart and other tissues, and also because it may suddenly shoot up to 106° or 107° , and even to 110° , and quickly cause the death of the patient. It is a good plan in typhoid cases to draw a line across the chart in red ink at the level of 104° , as a warning signal, to those who are watching the case, of the danger which may occur when the temperature reaches this height.

When you arrive at the conclusion that you have to deal with hyper-pyrexia, the next point is, what is to be done to reduce it? I shall suppose that you are dealing with a case in private practice, where the resources and assistance obtainable in a hospital are not at hand. In such a case you should commence treatment by sponging the patient from head to foot with iced water. He should be stripped and placed on a mackintosh sheet, and

the sponging be repeated every four hours, or even more often, until the temperature has been reduced to 102° or thereabout. Mere sponging, however, sometimes fails to produce the desired result, and then the next resort should be an ice-pack. The patient having been stripped and placed on a mackintosh sheet, an ordinary cotton sheet, folded so as to have four layers, is wrung out in iced water, and the interspaces between the layers should be packed with pounded ice. This pack is wrapped round the body, with the limbs left free, in such a way that the axillæ are left accessible for thermometrical observation. I may say that after many years' experience I have never known any harm result from this ice-pack, and feel convinced that there is no danger whatever of pleurisy, pericarditis, or pneumonia, etc., occurring from its use. In the case of the boy upstairs, whose temperature remained at 105° for 32 hours from the time of admission, he was ice-packed three times with, as you know, the happiest results. His case served to illustrate the course of the temperature under this application: the first pack steadied it, that is to say, it prevented a further rise; the second reduced it somewhat, but only for a time; and the third brought it down beneath the danger line permanently. This is the ordinary course of events. As with sponging, so with the ice-pack: you must not try to reduce the temperature much below 102° . Typhoid fever is a disease which necessitates a certain height of temperature, and a persistence with cold applications until the temperature is reduced to normal, or beyond it, is in my opinion not only useless, but harmful, and it masks the severity of the disease and entails grave risks to the patient. On each occasion the patient should be left in the pack until the temperature is 102° – 103° , and he may be kept therein even for hours if necessary. To illustrate how frequently the packing may be done, I will quote a case recently reported by myself.

"In the evening his temperature rose to 104.8° . He was constantly packed for 24 hours until the temperature was reduced to 102.4° , and on its rising to or above 103.8° this was repeated for the next seven days; after which he gradually improved.

Here was a case in which the patient was packed almost constantly for eight days. Surely if the procedure were ever attended by the dangers imagined by some, we should have had them in this case, which was an exceptionally severe one. Again, the boy upstairs was packed three times for some hours, and, as you have seen, there were no bad results.

Immersion in a bath is, I quite believe, a most useful procedure, but in private practice it is almost an impossibility, and I need not, therefore, discuss its value in a clinical lecture.

We now come to the question of antipyretics, and here, perhaps, I take a somewhat different standpoint to many teachers. I have given 20 grains of the Sulphate of Quinine at one dose in a case of rapidly rising temperature in a case of hyper-pyrexia without any result. Small doses, we are all agreed, are useless, and as large doses are unreliable I am opposed to their use. My experiences with Antipyrin and the many other febrifuge medicines now in use, have taught me to view their routine use as antipyretics with grave suspicion. A further objection to their regular use, in my opinion, is that even when they are successful in lowering the temperature, they are almost always equally depressant to the patients. Cold sponging or the ice-pack are both reliable means of treating hyper-pyrexia without exhausting the patient, and I prefer to rely on them rather than on drugs.

The points then that I wish you to remember are that when the temperature has remained over 104° —the danger line—for four hours, you have a hyper-pyrexia which must be dealt with, and that, in my opinion, the best method of reducing it is by either cold sponging or the ice-pack, and that when you have reduced the temperature to 102° or a little lower, you need not persist in these applications until the temperature again rises, and shows a tendency to rise still higher.

The next point to consider is the diarrhoea. It is well to remember that though diarrhoea is usually present, it is not always so; and also that as a rule, except it be aggravated by irritating food or drugs, it will nearly always cease without any treatment. With regard to diarrhoea as the result of irritating food, I will refer to it later when I speak on diet. As to diarrhoea from irritating drugs, that should not often occur in your practice, for I have rarely omitted an opportunity of warning you against the administration of purgatives in either the earlier or later stages of this disease. If the

diarrhoea is so great as to be exhausting to the patient's strength, and the patient is on an almost exclusively milk diet, you should first examine the stools for the presence of milk curds, and if these are found to be present, alter the diet to beef-tea and to milk, well diluted with lime or soda (not merely carbonated) water, and give small doses of Bismuth. Absence of further irritation will cause the diarrhoea to stop, or to become very slight in amount. In certain stages of typhoid the stools are always soft and characteristically pultaceous. I very rarely have had recourse to Starch and Opium enemata, except merely for transient relief so as to conserve the patient's strength, and when on rare occasions I have used them deliberately to arrest the diarrhoea, I have always had to repent their use. Think what this powerful checking of the diarrhoea means. We have examined together the stools of the two cases upstairs, and seen them to consist of pultaceous fermenting material. If you lock up in the intestine such fermenting material, the fermentation process must go on, gases will accumulate in the intestines, and you will get that dreaded complication—tyimpanites; and this may necessitate the use of the long tube, or even the tapping the intestine with a fine needle or trocar. I do not say such a condition of things will always occur, but there is a grave risk of it, and I would advise you, therefore, never to be in too great a hurry to adopt any powerful astringent treatment for the looseness or diarrhoea, but always try every simple and rational means first.

Whilst speaking on diarrhoea, I would remind you that there is no definite relation between its amount and the extent of ulceration of the intestine. I show you here an interesting specimen of a portion of large intestine, in which, as you can see, *all* the solitary glands, as well as the Peyerian patches in the small intestine, have been affected in the course of an attack of enteric fever. Yet, in the case from which this was obtained, diarrhoea ceased on the 10th day, and the patient was sitting up on the 18th day reading a novel. No diarrhoea re-occurred, but the patient eventually died on the 31st day, as the result of thrombosis of the femoral veins.

Hæmorrhage is a serious and alarming complication, and there is much difference of opinion as to the best means of treating it. I cannot say that my experience of astringents has been at all satisfactory; nor do I see even theoretically how

they are supposed to act after having been absorbed into the blood when they are only occasionally useful in arresting superficial hæmorrhages, where they can be brought into direct contact with the bleeding vessels. I have lost but very few cases from hæmorrhage, and have always relied on the careful external application of cold by ice compresses on the abdomen and the internal administration of Opium, which serves to keep the patient's bowel quiet and at rest.

Perforation is almost always, if not absolutely always, fatal. I remember the case of a man who rallied from the primary effects of a perforation, but died a fortnight later, owing, as the post mortem revealed, to the giving way of the adhesions, which had at first saved his life. When perforation has occurred give Opium freely. I prefer one grain of solid Opium every four hours, taking, of course, the precaution of watching the pupils. I cannot recall one single case of recovery after undoubted perforation.

I will only further deal with the questions of diet and alcohol, leaving the numerous other important points to a future occasion.

Diet during the active stages.—The diet must essentially be fluid. I give milk fairly freely, that is, from two to three pints a day. It is most important to watch the stools for the presence of milk curds. Should they be found, dilute the milk with lime-water, one part of lime-water to two parts of milk. If the curds are still found, cut off the milk almost altogether, and keep your patient mainly on beef-tea. I prefer using properly home-made beef-tea to any of the many preparations of beef now in vogue. In addition to the milk I give usually one pint of beef-tea per diem. In feeding cases of typhoid fever you should give food in small quantities, and at short intervals; thus, suppose that your patient is taking about two pints of milk, and about one pint of beef-tea in the twenty-four hours, that is to say, about 60 ounces of fluid altogether, you should give a little over four ounces every two hours, or a little over eight ounces every four hours, or where there is great prostration two ounces every hour. If the patient is asleep instruct the nurse to wake and feed him at the proper time. This waking is of no great consequence, for as a rule, it is incomplete, and he will soon be asleep again. Never be content with verbal instructions as to diet, but write every item down on paper, and let the nurse tick them off on each administration of food.

Diet for period of convalescence.—This is one of the most difficult and important questions in the treatment of typhoid cases, for as soon as patients feel at all better they are seized with an intense craving for food. You cannot proceed with too great care, or watch the effects of the food too assiduously. My usual method is as follows: after the temperature has been normal for ten to fourteen days, I give them in their milk some bread which has been mashed into a pulp; if this does not disagree, it is continued for three days, when they are allowed to have a little custard; all going well, the quantity is gradually increased, and at the end of another three days a lightly boiled new-laid egg is allowed; in another three days some boiled white fish is given, and after a few more days some meat is allowed, and so they gradually return to ordinary diet. At the least untoward symptom, such as diarrhoea or rise of temperature, they are at once put back on milk and beef-tea. You will see then that I feel my way very gradually, taking from two to three weeks for the patient to return to ordinary diet. I would impress upon you the great importance of this; in one of our cases upstairs, though now in the seventh week, the temperature, which had been normal for eight days, went up three hours after he had taken some bread pulp in the milk; and in the case of the woman we had to make three separate attempts before she was able to continue taking the diet I have just sketched out to you without a rise of temperature.

Lastly, we come to the vexed question of the use of alcohol. It always has been, and always will be, a vexed question, owing to the fact of the great social importance attached to it. There are some who consider that to use alcohol, in what is considered an attractive form, is to run the risk of leading people to drink. I quite agree that in some cases there is this risk, but this is in cases where alcohol is given for fatigue or neuralgic pains, or for a large number of those minor conditions in which temporary comfort leads to frequent "nipping," until such patients become its slaves, and believe, or pretend to believe, that they cannot exist without it. Careless ordering of alcohol for such cases is more than reprehensible; but I would submit that there is no analogy between these minor complaints and grave cases of typhoid. In the former, the patient is fully conscious of his actions, and always has the alcohol at his service; while in the latter, the patient is in anything but a

conscious condition, and the alcohol is given him, and taken by him, as any ordinary medicine, and as such it should be regarded. There is no mental exaltation, no special sense of physical comfort after its administration, but it serves to keep up the failing heart, which is all that is aimed at. I am aware that my views are not orthodox, but they are forced on me by many years of practical experience.

Alcohol, of course, must not be given haphazard in all cases, but only where there is special indication, and then it must be given freely. The chief indications on which I prescribe it are :

- (1) A weak first sound of the heart ;
- (2) Dicrotic pulse ;
- (3) Wandering delirium ;
- (4) Involuntary passage of fæces and urine.

The late Dr. Murchison taught that 8 oz. per diem of brandy was always enough ; and that, if 12 oz. per diem did not save the patient no further amount would. For cases of moderate severity no doubt this amount is ample, but there are some cases in which much more must be given, and in which I feel sure that a much freer use has saved the patient's life. I will relate to you, briefly, two typical cases in which I believe a fatal termination must have taken place but for the free exhibition of alcohol.

Case 1. His condition from October 9–15 is thus described in the case-book : " Patient lies quite unconscious ; face blue ; subsultus very marked ; diarrhoea continues, and occasionally a little blood in the motions, which, as well as the urine, are passed involuntarily. Tongue black and dry ; pulse 144, almost imperceptible. On the 15th he began to show some signs of improvement, and from this date he slowly progressed towards recovery, although there was a relapse lasting 18 days. He was packed whenever the temperature rose above 103.5, and the daily quantity of stimulants given to him was : October 3rd, 4th and 5th, 10 oz. ; on the 6th and 7th, 15 oz. ; 8th, 15 oz. and two pints of champagne ; 9th to 14th, 20 oz. and two pints of champagne ; 15th, 15 oz. ; 16th, 10 oz. ; 17th to 22nd, 6 oz. ; 23rd to November 5th, 4 oz. ; and November 6th to 8th inclusive, 2 oz. ; making a total of 310 oz. of brandy and 4 pints of champagne."

Case 2. " On the 19th at 9 a.m., his temperature having risen to 104.4, he was packed, and this was renewed again and again when his temperature rose above 103.3. From September 17th

to 27th inclusive he was quite unconscious ; his pulse was 140 on the average, fluttering, and irregular. He had some diarrhoea. On the night of 22nd his temperature fell 8° (105–97) without any hæmorrhage, and every one expected his immediate death. By the persistent application of warmth and stimulants he rallied, and on the 27th he began to improve. A relapse occurred on October 12th, lasting till 26th. A second relapse on November 12th, lasting three days. He slowly convalesced, and was sent home to Norway Dec. 12th. Brandy, September 15th, 16th, 17th, 4 oz. ; 18th, 6 oz. ; 19th, 6 oz., and 2 pints of champagne ; 20th and 21st, 10 oz., 2 pints champagne ; 22nd and 23rd, 12 oz. ; 24th to 30th inclusive, 16 oz. ; from October 1st to 4th, 12 oz. ; from 5th to 8th, 8 oz., 2 pints champagne ; from 9th to 17th, 4 oz. and 1 pint champagne. On 18th brandy was raised to 6 oz., again reduced to 4 oz. on the 25th, when the champagne was stopped. On November 6th 2 oz. only were given daily, and on the 9th was discontinued. Total quantity consumed was 388 oz. of brandy (2 gall. 3 pt. 8 oz.) and 21 pint bottles of champagne.

A CLINICAL LECTURE

ON

TWO CASES OF THYROID TUMOUR

Delivered at St. Bartholomew's Hospital, Feb. 10, 1894,

By H. T. BUTLIN, F.R.C.S.,

Surgeon to the Hospital.

GENTLEMEN,—I have here four patients whom I wish you to examine for yourselves.

In the first case there is not much to see ; but I want you to study her by the light of this photograph. It is a case of myxedema, treated, not by the injection of thyroid juice, but by its administration. A comparison of her present condition with that at the time of her admission, as shown in the photograph then taken, is instructive.

The second patient had a thyroid cyst removed about a month and two or three days ago. I want you to see what kind of scar is left at the end of such a period. In course of time the scar will become quite white, and gradually less and less marked, until practically it is not very noticeable.

Of course there will always be some kind of scar.

As I give a description of the other two patients I want you to come down and examine the condition of the tumour in each case.

I put up a notice to say that I was to give to-day a clinical lecture on "Two Tumours of the Thyroid Gland." There is a great difference between the two. I take the first case first because it is the more interesting of the two, and it is the more rare.

The patient has some enlargement of the thyroid gland. The enlargement is greater in the two halves than in the centre, and much greater in the right lobe than in the left. It stands out in the form of little bosses or tumours. The gland is so firm in consistence as to feel absolutely hard, as if composed of calcareous matter. It does not move up and down freely, as does the lump in the other case. In the deeper parts it is quite fixed, but I do not think it is at all fixed to the tissues over or about it. From the feel of the tumour and the age of the patient I should judge that it must be either a goitre which was formerly fibrous, and has shrivelled up and become calcareous, or a carcinoma of the thyroid gland. My own feeling is rather in favour of its being a carcinoma on account of these lumps which protrude, and from the fact that the patient gives no previous history of having had goitre, and has only been aware of anything amiss in her neck for the last six months or thereabouts. In fact she was under the care of my friend Mr. Doran at the Samaritan Hospital for some trouble. She pointed out to him that there was some swelling in the neck; and he sent her to me as it was a matter which to him was not particularly interesting, but which he thought would be so to me. I no sooner saw her in the consulting room than I came to the conclusion that the case was unusual in perhaps more than one respect, and I took her into the hospital.

In addition to this lump in the thyroid gland, it struck not merely myself but others who saw her, that she had some symptoms which might possibly belong to myxœdema. Her hair is coarse, short, and not very thick; her features are not so clearly defined as they might be, and her lips are often of a livid or bluish colour. Again, the skin of the fore-arms is exceedingly rough. I thought this might be due to the nature of her work, but she tells me that she has no other work than her household duties, and she has done nothing that

would cause such roughness of the skin. Although she has now been in the hospital for a period of two weeks or more, and has done nothing to keep up the roughness of the skin, it still remains and has not improved. The skin of the body is, I think, a little drier than natural, though I do not know that there is very marked dryness, as at this season of the year there is but little perspiration.

At first consideration you might ask what, after all, there is so peculiar in the fact of a patient who has some disease of the thyroid gland having symptoms of myxœdema. Of course, if this is myxœdema it is in a comparatively early stage, and her history is probably perfectly correct that she has only had some affection of the thyroid gland for a few months.

In the first place, then, let me point out to you that if this is a case of carcinoma of the thyroid gland, it is an exceedingly rare case. During the last five years there have been, so far as I am aware, only two cases of malignant disease of the thyroid in this hospital. I have looked up the hospital statistics, and find only these two cases under the care of all the physicians and surgeons. Again, so far as I am aware, there is only one case on record in which myxœdema has been associated with malignant disease of the thyroid gland.* You know that myxœdema, as a rule, does not occur in hypertrophies of the thyroid gland; that, in fact, it is associated with atrophy of the gland, so that you can scarcely feel the thyroid gland in people who are myxœdematous. The name *cachexia strumipriva* has been given to the condition resulting from removal of the thyroid gland. After its removal or atrophy patients are liable to myxœdema, but only after removal of the entire gland. If a small part, or even what is called an accessory or supernumerary thyroid gland, is left behind, this appears to be quite sufficient to preserve the patient from all danger of myxœdema. If this is a case, as I believe, of malignant disease of the thyroid gland, carcinoma with symptoms of myxœdema, then I take it that the carcinoma must have destroyed the entire secreting structure of the gland; and that seems to me to be by no means improbable, because I cannot trace any healthy feeling in it.

The other patient is suffering from what I believe to be a cyst of the thyroid gland—a very much

* "Pathol. Soc. Trans.," vol. xxxvii. p. 511—Dr. Gulliver.

more common disease than the other. In the last three or four months I have had in this hospital two cases in which I have removed cysts of the thyroid gland; this I take to be a third. I have seen one outside the hospital as well. It is not, however, a very common disease. I think I have had rather more than my share of such cases.

I judge that this is a cyst of the thyroid gland, partly on account of its situation. It is just in and a little to one side of the isthmus; and that is the seat of election of thyroid cysts. Then, too, it is a circumscribed tumour, and there is no obvious general enlargement of the thyroid gland. It is more or less rounded. It appears to me to fluctuate; but I cannot be quite certain that it does so. It is difficult to be certain whether there is fluctuation in a tumour in this situation; but even if it is a cyst it may contain a certain quantity of solid matter, and a very small quantity of fluid, in which case it is more difficult to detect fluctuation. It is capable of being moved from side to side, in this respect differing from the tumour in the other case.

I have here two or three pictures of these cysts. One of them occupies a very high position in the neck. One of them is a picture of this patient. It has just been taken, but, unfortunately, is not a good one. Here is a cyst of the thyroid gland which I treated some time ago by injection.

This patient also, curiously enough, presents some symptoms which might possibly be classed as symptoms of Graves' disease. Her eyes are a little more prominent than, perhaps, they ought to be. Her friend, who came with her, told me she had noticed the patient's eyes were "more brilliant;" the fact being that they stand out a little more than usual. Her pulse is quicker than it ought to be. She has no enlargement of the heart. The heart-beat is practically regular. She has for some time past suffered from breathlessness, especially on walking up hill or going upstairs. She also complains of fulness of the abdomen, and it has, in fact, become somewhat fuller or bigger, so that she has had to let out her dresses. These, you will remember, are some of the symptoms of Graves' disease. I use the word Graves' disease instead of "exophthalmic goitre" because, although a goitre is frequently present in Graves' disease, and, in fact, is supposed to belong to it, there are cases of Graves' or Basedow's disease in which there is no appreciable enlargement of the thyroid gland. I showed these

patients to Mr. Berry with great pride, because he takes a very great interest in diseases of the thyroid gland, and has delivered some most excellent lectures on the subject; and he is of my opinion that the first patient presents some symptoms of myxœdema, and the other is suffering from Graves' disease, although she has not the most marked symptoms of it; but he does not think the lump in the thyroid has anything to do with it. The kind of goitre which is associated with Graves' disease is usually a uniform enlargement of the thyroid gland, a soft and pulsating enlargement; this patient, so far as I can feel, has no enlargement of the thyroid gland at all. My own impression is that if this lump has anything to do with her symptoms of Graves' disease it is only indirectly, just as some other conditions may have, possibly, to do with the production of the disease.

What becomes of these cysts of the thyroid gland? Well, I suppose it is possible that a cyst here may become gradually smaller until it, so to speak, shrivels up and disappears. On the other hand, it may grow larger; while, again, it may remain stationary, and that for many years. I cannot say that I have ever met with a case in which the cyst has disappeared or got smaller. I have seen several cases in which it has remained about the same size, presumably, for a long period of time; but the usual course of these cysts or cystic tumours of the thyroid gland is to grow larger and larger; and as they do so they produce symptoms which are more or less distressing to the patient. They produce pressure on the adjacent parts, and so, perhaps, push the larynx and trachea over to one side, or if the cyst remains fixed in the middle line, it may, by its pressure on the trachea, bulge it in, and cause some stridor in the breathing, or even, where it is of large size, produce very considerable difficulty of breathing, so that patients have come to me, not on account of the lump, but because of the dyspnoea.

Before I enter upon the question of the treatment of these cysts, I want to direct your attention for one moment to the *nomenclature* of the disease. I have for years been in the habit of speaking of these tumours as cysts of the thyroid gland; and I have done so whether the cyst was simple or compound, whether filled solely with fluid or containing a certain quantity of solid matter. I have not looked at the literature of the subject for a long time until lately; but the other day on looking it

up I found that these cysts are placed under the general heading of "cystic goitre," or "cystic thyroid." On going over one of these cases in the ward the other day I found that nearly all my clinical class after examining the patient said she was suffering from "adenoma of the thyroid gland." I thought, of course, that they had all taken the tumour to be a solid one; and as there was a good deal of doubt as to whether the tumour was fluid or solid, I was a little astonished at the unanimous opinion that it was solid, as indicated by the name given it. Afterwards I discovered that those who thought it adenoma still considered it a fluid tumour, or one containing a certain quantity of fluid. But Mr. Berry, I think it was, had been over the case with the Fellowship class, and had expressed his opinion that it was an "adenoma," on the ground that these cysts, according to his theory and the specimens he has, are really developed from adenomatous tumours—that the cyst is, so to speak, a mere accident in association with a solid tumour. I saw Mr. Berry afterwards and spoke to him about this, when he told me that he has a number of sections which prove that these cysts are developed from gland tissue, and that the gland tissue surrounds the cysts, and is really the essential part of the tumour. He told me that in all probability when I came to remove the tumour, I should find a quantity of solid growth around it. I did not do so. I removed a simple cyst, clearing it out as closely as I could with my finger, leaving absolutely no solid material behind. Again, within the last year I saw a lady who had consulted me two years previously about a large tumour which I took to be a cyst of the thyroid gland. I advised her on the first occasion, nearly three years ago, to have the cyst tapped and injected. Behind it, I may say, she had a chain of enlarged glands. The cyst was of considerable size, being bigger than my fist, and produced such discomfort, stridor of the breathing, etc., that she could not take active exercise, or ride on horseback, to which recreation she was devoted. A year ago she came to me a second time with the cyst considerably larger. On the first occasion she had been accompanied by her husband; the second time she came in deep mourning, having meanwhile become a widow, so that she had now plenty of time to attend to her own troubles. I ventured on that occasion to suggest that the cyst should be not injected, but removed. She was a

very cautious lady indeed, and very intelligent, and asked me a great many questions, the majority of which I was able to answer, but some of which I could not. She wanted to know why I had changed my opinion with regard to the method of treatment. I explained that the tumour was much larger than it had been, and it would probably be safer to remove it. I think she had been left in comfortable circumstances, for she seemed disinclined to submit to any treatment which might possibly cut short her existence. She balanced her present condition against the chance of my doing some damage by operation. In order to make quite sure she consulted several distinguished surgeons. One of these gentlemen, not knowing the antecedents of the case, thought it was quite possible that the disease might be malignant on account of the chain of glands; but I knew these had been enlarged for two years, so I did not attribute much importance to their enlargement. Another surgeon wrote to me to this effect:—

"My Dear BUTLIN,

"I think the disease from which Mrs. — is suffering, is in great part a cystic thyroid. I should not recommend, unless her life is imperilled, that she should run the risk of operation."

However, with great difficulty I persuaded her to have the tumour removed, and I took out from the neighbouring structures, not a cystic thyroid, but a large cyst like a bladder. There are three different names for the same thing. I call them cysts of the thyroid, Mr. Berry calls them, I suppose, cystic adenomata, and this gentleman who wrote to me called it cystic goitre or cystic thyroid. After all, you may say, what does it matter if you all mean the same thing? What does it matter whether you speak of thyroid cyst or cystic thyroid? I think in this case it does matter very much, because my impression is that the name may produce a very considerable influence on the opinion as to the kind of disease or its exact nature, and may quite possibly influence the treatment. I believe, for instance, in the case of that lady, when the surgeon who wrote to me said, "I think it is a cystic thyroid," he had in his mind a good deal of solid growth about, and in addition to the cyst; and I think, he took very rightly, a much graver view of the operation than I did, and was not inclined to recommend it. Again, if I took the view that the essential part of these tumours is adenomatous,

I should feel bound to remove a more or less considerable area of solid growth around the cyst. I did not do so; and yet I have seen these patients years after the operation in which the cyst has been shelled out, without the removal of any solid growth at all, and there has been no recurrence and no thickening, so that one is led to think no solid growth has been left behind. In these cases, therefore, I strongly recommend you to use the term "thyroid cyst" or "cyst of the thyroid," and to do so whether the cyst is simple or compound, fluid or solid.

We now come to the question of *treatment*. Let me tell you, with regard to malignant disease, about this specimen in which you see what mischief may be done by it. There is a great mass breaking down. The patient discovered one morning what at first was considered a cyst, but which soon showed itself to be malignant. The disease made rapid strides, and in six weeks or two months from its first discovery he was dead. Here is another specimen from a case in which could be felt this kind of lumpy growth. I do not know how long that patient lived, or what the exact cause of death was, but I think you will see sufficient disease to have produced a fatal result.

I could give you half-a-dozen different ways of treating thyroid cysts, but I shall limit myself to two, partly on account of our limited time and partly because really to my mind there are only two methods of treatment which are generally applicable to these cases. In the first place you may inject the cyst, in the second you may remove it.

The injection has for its object to produce suppuration. You inject into the cyst some stimulating or irritating fluid, which shall, in fact, change the cyst into a chronic abscess. In time, the suppuration becomes less in quantity; and in due course, if all goes well, the cavity completely contracts until nothing but a tiny sinus is left, and, by and by, the patient is cured.

Instead of giving you the book account of it, or such a description as Sir Morell Mackenzie has given,—for it was he who reduced this operation to a method,—let me describe the first case I treated in this way. Ten years ago a very anæmic, delicate-looking lady was sent to me with a lump which I took to be a thyroid cyst. It was in the isthmus of the gland. I deemed it best to inject the swelling, and not remove it, as she did not wish to have a scar. Accordingly in the next year,—for

she was not well enough to undergo treatment when I first saw her,—I gave her a little gas, and using a fine trocar and cannula, of which the plate is pierced for silk thread, and which is made for the purpose, I thrust it into the interior of the cyst. I drew off some brownish fluid,—not so much as I expected,—but did not empty the cyst. There came through the cannula some solid brownish substance, which I took to be altered blood-clot. It may have been so, on the other hand it may have been some intracystic growth. Then I injected a solution composed of two drachms of Tincture of Perchloride of Iron to the ounce of water into the cavity, and fastening the thread round the neck I put into the hole in the cannula a little plug. This was left for three days, at the end of which the plug was taken out, and a very little brownish fluid came out through the cannula. Poultices were then applied all over the neck. Every day the plug was taken out, but the cannula was left in the swelling. In the course of a week or ten days the swelling had become very hard and tender; and at the end of ten days or a fortnight there came out, not brown fluid, but pus. As soon as suppuration was thoroughly established the patient went to the country, so that I only saw her at comparatively long intervals of a month or six weeks. Every time she came in I passed a probe to see how far from the end of the cannula I could push it. At first I could introduce it for an inch or more, but gradually the distance became less until I could not push it beyond the end of the cannula. The suppuration during this time became less, and the swelling of the neck decreased. I took the cannula out, and in the course of three or four days the opening healed, and the patient was cured. The cure in that case occupied six months; and as I could not take the cannula out, although she was able to go about and do what she wanted to do, she could not go into society; but was obliged to limit herself to walks and simple recreation. It really took six months to complete the cure of a cyst by injection of Perchloride of Iron. This fluid was chosen and strongly recommended by Mackenzie on account of its hæmostatic properties, for some of these cysts are apt to bleed, sometimes very seriously.

The advantages of the method are that there is no "operation," it can be done without an anæsthetic, and if it is successful it leaves no very apparent scar. About seven years after the treatment had been concluded, I saw the lady whose

case I have just described, and she was perfectly well. She has never had any recurrence of the tumour in the neck; and the scar had to be searched for, as it could only be seen with difficulty. You will notice the difference in this respect between her case and that of the young woman you have examined. The scar in this young woman's case will be white, and the marks left by the sutures will almost disappear; but there will always be a scar.

Let me now give you an account of the other method of treatment—that which I shall adopt to-morrow for the other case—the method of removal by what is called enucleation. I have removed a good many of these tumours, and for the last three or four years I have adopted always the same procedure. I expect to cut down on the anterior border of the sterno-mastoid, or possibly a little nearer the middle line. If the incision is on the anterior border of the sterno-mastoid there is less obvious scar. I shall cut through the skin, the platysma myoides, and the deep fascia. Then I shall come upon what requires to be very carefully distinguished and separated from the cyst, namely, the capsule of the isthmus of the thyroid gland, and underneath that a thin layer of the isthmus itself of a brownish colour. It is most important to distinguish between that and the cyst, because if you attempt to separate the isthmus, you come down outside the lobe of the thyroid gland, and may get into serious trouble. The thin layer of isthmus requires to be divided, and I shall do so without any hesitation, as I do not expect any serious bleeding from it. Then I hope to come down upon the cyst wall itself. That I expect to distinguish by its bluer colour, its more fibrous aspect, and possibly a certain translucence. The one is of a brownish colour, has a solid appearance, and is beautifully smooth; the other is almost equally smooth, but has a more fibrous texture and a bluer colour, and may be translucent. I shall divide all the structures over the cyst as far as necessary for its removal. I shall then do my best to remove the cyst entirely, without using either knife or scissors for the rest of the operation; and I daresay I shall be able to do it with my finger. I may possibly have to make a cut here and there with the blunt end of the scissors, where a band of fibrous tissue or fascia adheres closely to the surface of the tumour. It is of great importance to avoid wounding the tumour; and fortunately most of these cysts are so thick-walled, that there is very little danger of this. If

by chance I should do so, I shall fasten up the opening with a pair of clamp forceps, so as to keep the cyst as whole as possible during the operation, and deal with it as a large rounded body. In one of my early operations where I was unfortunate enough to cut into the cyst, and in which the cyst was thin-walled, I did certainly get out the cyst after a certain time, but the patient's voice was for some time not so good as before, and on examination one of her vocal cords was seen to be immobile. She recovered her voice completely, but the vocal cord never recovered its movement. Every patient, however, will not recover her voice so well as that one in these circumstances.

The operation of removal by enucleation is suitable for people who are anxious to get the treatment over as soon as possible—people who do not mind a scar. The majority of hospital patients really are indifferent to such a scar, provided the treatment is quickly over. Again, the cure is much more certain. If there is a large quantity of solid material in the cyst, the probability is that you will not cure it by injection. A small quantity of intracystic growth does not, however, I think, interfere with the treatment by injection. I have seen several cases in which I suspected some solid growth and which I injected; and in the course of time they were completely cured. Of course, if the injection treatment fails, the treatment by removal can be carried out.

At the present time there is a kind of fashion for treating these tumours by removal. In the medical papers every now and then, or in the transactions of various societies, you will see papers by this or that surgeon strongly recommending the removal of these thyroid tumours and cysts in preference to injection or other procedure. Our fashions in these things, like many other fashions, are some of them good and some not. My own impression is that the treatment by injection—although I have not carried it out for three years—is well adapted for a certain number of cases. If a young lady, belonging to the fashionable world and going into society, were brought to me by her mother suffering from this condition, and I said to her: There are two ways in which this can be treated; the first is by removal, after which your daughter will be well in a few days, with little risk, but there will always remain a mark; the second is much longer, not so certain, entailing the production of an abscess for which she will have to wear an apparatus for, say, six months, at the end

of which, however, there will be practically no scar; even if I were to say that the mortality in the one case was 4, in the other 25, I am pretty certain the lady would desire the injection method.

If the cyst is small; if it is single; if the patient is in good health, and suppuration is not of importance, and time is a matter of no consequence, while on the other hand the production of a scar is a matter of great consequence, you may fairly choose the treatment by injection. Treatment by removal is applicable in almost every other case.

As to the relative dangers of the two methods, the treatment by removal has grown into favour a great deal, I feel quite sure, partly on account of dangers and deaths which have occurred every now and then in the treatment by injection. At the very moment the fluid has been injected, in some cases the voice has been lost, sometimes permanently. Even sudden death has occurred, presumably through clotting of the blood in one of the large veins connected with the thyroid. On the other hand, you will every now and then read papers by men who tell you they have done 20, 30, or 40 removals by enucleation, and not one of the patients has been seriously ill, not one has suppurated, and certainly not one has died. The picture is a little too good. In my own experience I have never seen any serious consequence from injection. But I have lost one patient after removal of a cyst. He was an old man of 70, with an enormous cyst. I obtained the opinion of my colleagues as to the advisability of dealing with it. He had it, I think, for thirty years, and it had grown to a large size, until it pressed his trachea and larynx towards the right side. He had immobility of the left side of his larynx, stridor and severe dyspnoea at times, so that I was obliged to urge upon him the desirability of operation, as I considered his life in urgent peril. I removed the tumour as I have described. There was some hæmorrhage at the end of the operation, but not severe. He was taken back to the ward, but he soon developed Cheyne-Stoke's breathing, and in the course of two or three hours died. I do not know at this moment what he died of. I certainly did not cut or damage any deep structures in the neck; but I suppose in the course of time this old man's tumour had become an integral part of his system, so that its sudden removal disturbed the vital processes.

I looked up the other day the accounts given

of the removal of a number of these cysts, and found that two patients had died of hæmorrhage. I myself have had a case of hæmorrhage after removal, but a very slight one. I, therefore, cannot help thinking that there are more deaths after removal of these cysts than we are at first inclined to admit. I should think, in all probability, the mortality after removal, although very small, is, on the whole, somewhat greater than the mortality after injection.

You may ask whether it is a desirable thing to remove a cyst or tumour from a woman presumably suffering from the symptoms of Graves' disease. She has no hypertrophy of the heart, certainly, but still she has symptoms of nervous disease, and you may see she is a very nervous and excitable woman. On that question I am quite sure. It is quite possible the removal of her tumour may cure her of her Graves' disease, if that is the disease from which she is suffering. You know Graves' disease is regarded by some people as a nerve disease, and the enlargement of the thyroid gland is regarded as part of the effect of the nerve disease, perhaps of disease of the sympathetic system. Now, various mental troubles have the reputation of leading to, or producing, Graves' disease; but it is, on the other hand, well known that patients get well with the exhibition of various medicines, with rest, etc. Within the circle of my own family, an illustration of this was afforded where the patient was entirely cured by perfect quiet for a period of some months; and that was a good many years ago. It has become very fashionable to remove a large part of the thyroid gland for Graves' disease, and it is said cases so treated recover; but, on the other hand, there have been cases where slight nasal obstruction has existed with Graves' disease, and the application of the galvano-cautery to the swollen tissues has been followed by complete recovery, so that the larger operation of the removal of a large part of the thyroid gland might have been unnecessary. The patient might have got well by some quite simple operation. Some mental shock or something which would have affected the mind of the patient might possibly have produced a cure. In this case the removal of a small tumour, which is very desirable for other reasons, may possibly be followed by improvement in her general health; and I should not be in the least surprised if I, what is called, "cured" her of her Graves' disease by the removal of this tumour.

STRETCHING the SPHINCTER ANI.

BY

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IN July, 1893, I contributed an article to the "Clinical Journal" entitled "Clinical Remarks on the Treatment of Piles." The article was a concise account of the method of treating piles, which I have employed in my hospital and private practice for some years, and was written as nearly as possible in the words of a clinical lecture which I had delivered about that time. In that article I referred to stretching the sphincter ani as a usual and necessary preliminary to the operation of ligaturing piles, which is now my almost invariable practice. I also described the manner in which the sphincter is dilated.

On the 20th of December, 1893, there was published in the "Clinical Journal" a most able and interesting clinical lecture by Mr. F. Treves, F.R.C.S., on "Hæmorrhoids." Near the end of this lecture, in remarking shortly on various methods of treatment, Mr. Treves says "(3) Dilatation of the sphincter with bougies is not of very much value. It has been assumed to act by the rest that is necessary, both before and after the bougie is passed. A large bougie, No. 12, is used. How it acts it is difficult to say."

I was much disappointed on reading this paragraph at the time, because it is so inaccurate and misleading, being, therefore, most unlike Mr. Treves' usual statements. Dr. Clifford Allbutt has very properly (in the number of the "Clinical Journal" for 24th of January, 1894,) drawn attention to Mr. Treves' mistake, and has ventured to say a word in favour of the usefulness of dilating the sphincter in operations on the anus. Having employed this method for several years as a regular part of my practice, I desire to say something also in its favour.

My attention was first drawn to the benefits that are derived from dilating the sphincter by an article I read in an American journal in 1884, and my mind having been previously prepared by a thorough and experienced belief in the doctrine of *rest* as taught by Hilton, I at once adopted the practice, and have had good reason to be satisfied with it in every way.

Hilton says (page 275, Second Edition of

"Rest and Pain," 1877), in speaking of the cause of the great and persistent pain associated with *fissure of the anus*, "the cause of the continued painful contraction which accompanies it lies in the *enduring strength* (italics mine) of the sphincter muscle." Further on he says, "Therein lies the *rationale* of the operation so frequently performed (division of the sphincter as recommended by Syme long ago), it prevents the muscle irritating or annoying the surface or edges of the ulcer by pressing them upon each other during its contractions. . . . The treatment of such cases is really absolutely based upon bringing about local physiological rest, for, by dividing the muscular fibre you merely prevent further friction by the contraction of the muscle." Mr. Hilton also remarks, that in cases of long-standing trouble from fissure one finds the sphincter "hypertrophied, massive, and increased in dimensions." He might have added, increased in power, for most surgeons know from experience the power of the sphincter in cases of well-marked painful fissure of the anus, to oppose the entrance of the surgeon's finger and to compress it if he manages to get it inserted.

"It is worthy of notice" (continues Mr. Hilton) "that when the muscle has been divided in such cases, it soon returns to its more natural condition, by the muscular fibres resuming their natural dimensions."

Now, what Mr. Hilton states with regard to the operation of division of the sphincter, I claim for the less severe and more generally applicable procedure of *stretching*. And what Mr. Hilton says of the pain-producing power of the sphincter in cases of fissure of the anus applies also to the power of the same muscle to produce pain in cases of piles especially when they are inflamed, and after they have been operated on.

My desire in writing the present article is not merely to advocate the importance of stretching the sphincter in operations for piles, but to point out the general applicability of the procedure to most operations in the neighbourhood of the anus.

To begin with the *modus operandi*. The proper and only safe way of stretching the sphincter ani is by the hands; in this way only can one know with any exactness what one is doing. Dilatation by a speculum can not be done so safely or accurately or intelligently, and the use of the bougie, it seems to me, can only do harm and no good. How a bougie passing in can act otherwise than a

mass of fæces passing out, I cannot see. And it is well known that the passage of fæces does harm in piles as well as in fissure. The digits that should be employed in stretching are the thumbs.

Preparatory to the performance the patient ought to be fully anæsthetized. This is absolutely necessary for several reasons. (1) The operation is both disagreeable and painful. (2) I have seen considerable shock occur when the patient was not "fully under." (3) If the sphincter is hypertrophied, as described by Mr. Hilton, it is not easy to overcome it while the patient retains consciousness and power. I have never used Cocaine. The patient being fully under the influence of the anæsthetic, one thumb, well oiled (for this and similar purposes I use Vaseline with 10 per cent. of Cocaine and Eucalyptus), is inserted well into the rectum, then the other thumb is inserted also, the two being back to back, and then they are separated in an antero-posterior direction, gently, slowly, but firmly till the fibres of the sphincter are felt to give way and the muscle no longer resists. A separation of something like two inches between the thumbs is generally necessary. But the real and only safe and certain test, is the complete flaccidity and loss of contractile power (for the time being) of the muscle. In some cases the muscle gives way easily, in others considerable force is required, but in no case should a sudden or rapid tearing be attempted. The result aimed at is *paralysis*. That is to say, the contracting power of the muscle must be, for the time, suspended, the object of the whole procedure being to give the surgeon ready access to the lower part of the rectum, and secondly, to prevent the painful contraction of the sphincter after the operation, which is the cause of so much suffering to the patient—or used to be. It is not difficult for the operator to make certain of the exact point at which he has attained to the abolition of resistance. Whenever the anus is quite flaccid and patulous, then the desired result has been obtained.

During the stretching there is a distinct perception of tearing, which is rather unpleasant to the operator, especially the first time it is experienced. I have never seen any harm result in the way of laceration, or subsequent ecchymosis. When the dilatation is complete, the lower inch or so of the rectum immediately protrudes on the removal of the thumbs. This also may be a surprise to the operator when experienced for the first time; but it is a great assistance afterwards if piles have

to be dealt with, because it generally saves the necessity for a speculum, and certainly removes all occasion for dragging down the piles into view. An equally thorough view of the lower part of the rectum cannot be obtained by anæsthesia alone.

The after-effect of this operation of stretching is, however, the most important—certainly from the patient's point of view; viz., prevention of the spasmodic contraction of the sphincter, which causes so much suffering in cases of fissure and piles.

I remember well the days of suffering that I witnessed when I was a student, and in the first years of my own practice. The poor patients were told that it was inevitable, and that they must just grin and bear it, or perhaps the burning agony was mitigated by Morphia suppositories by which they were stupefied. Since I have commenced to employ dilatation of the sphincter, I have hardly ever used Morphia; my patients have been quite comfortable, and have complained of nothing but of having to lie in bed while they felt quite well, and of being kept on low diet. Indeed, I think that, had I the courage of experience, I would let them get up and go about. But I don't like to experiment. Furthermore, my patients have always slept well, and I do not remember having had to use a catheter.

There are no bad after-consequences that I have observed. I have already said that I have not seen any undue laceration of skin or mucous membrane. In a case of fissure the ulcer may be torn, but there is no harm in that. It is only copying what is done by incision. Neither have I seen any extravasations. Incontinence of fæces should not occur. In the first place the rectum is thoroughly emptied, and should be kept empty. In the second place the sphincter recovers its power wonderfully soon.

There is no true paralysis produced. The muscle begins to recover its function at once, apparently. A certain amount of power is perceptible before the patient leaves the operation table. I have even had to repeat the stretching in an operation that took somewhat longer than usual to perform. Patients are conscious of being able to retain flatus soon after they come out of the chloroform; and by the time it is necessary to have the bowels acted on complete power of the sphincter has returned. While investigating the condition of the anus day by day it is possible to ascertain by the tip of the finger, used gently, that

the sphincter is soft at first, but active enough, and that its power returns gradually but surely. Complete normal power and function return to [the sphincter in from one to five days—according to my experience.

This method of procedure is essentially in the lines of Mr. Hilton's teaching. It is procuring rest—physiological rest, as he called it.

I have noticed such an enormous difference produced in the condition of my patients—a change from burning, agonizing suffering, lasting often for several days, to comparative comfort, without any risk whatever, that I would almost use the language employed by some when antiseptics were first introduced, that not to employ the means would be criminal.

I wish now to draw attention to the general applicability of stretching of the sphincter in the treatment of affections of the anus. Let me take *fissure* first—Hilton's typical painful disease. Hilton has proved that the pain in this disease is caused by the contractions of the sphincter; the ulcer is kept from healing mainly by the periodical splitting up that takes place every time the bowels act. By thoroughly stretching, and for a time paralyzing the sphincter, the source of pain is abolished, and the base of the ulcer is torn, in which, as Mr. Hilton has pointed out, there often is an exposed nerve filament. With rest and perfect cleanliness the ulcer ought to heal in a few days. When the bowels act after this there ought to be no suffering, because the ulcer ought to be healed, the sphincter in the interval having lost the spasmodic contracting power that caused the suffering, and prevented healing of the ulcer.

My usual procedure in cases of fissure of the anus, uncomplicated by piles, is to stretch the sphincter thoroughly, to cut off the tag of skin that is usually at the root (perhaps in more senses than one) of the fissure, to keep the patient in bed for one day, and then to let him (more frequently her) go about as usual, only prescribing a limited diet, perfect cleanliness of the anus, and a laxative on the second or third day. This method of treating a fissure of the anus, I consider, is a long way in advance of any other that I know of, and is probably perfect.

Let me now refer to *fistula*. Syme pointed out long ago that the constant action of the sphincter might interfere with the spontaneous cure of a fistulous track, and that slitting up

the fistula by cutting through the sphincter probably acted as much by securing rest as by enabling the resulting wound "to heal from the bottom upwards." Whether this idea be correct or not, paralyzing the sphincter may be of use in two ways in the treatment of fistula. If done before cutting the fistula open it makes the operation more easy and prevents the possibility of any spasmodic contraction of the opposite half of the muscle taking place after the operation. Another use (which I have not tried, however) might be to paralyze the muscle in nervous persons who are afraid of being cut, or in busy persons who cannot afford to lay up for a few days. The thing might be tried, and could do no harm.

I now come to what may be called the disputed point, viz., the use of stretching the sphincter in the treatment of piles.

To put the thing briefly, I would say (1) Stretching the sphincter makes access to the lower part of the rectum easy. (2) It prevents spasmodic contraction of the sphincter after the operation.

Mr. Treves says that dilatation by bougies is not of much value,—in that I agree,—but I hold that stretching by the thumbs is of great value. Mr. Treves also says, "How it acts is difficult to say." How passing bougies would act beneficially is, I admit, very difficult to say or understand; but how stretching by the thumbs acts beneficially is abundantly plain, as explained by Mr. Hilton.

Stretching the sphincter is specially useful, I think, in the operation by ligature because that operation is most likely, of all the methods ordinarily employed, to be followed by painful spasmodic contraction of the sphincter ani. But stretching is, I am sure, of much wider applicability. I have employed stretching with success in the following conditions.

(1) In cases of inflamed external piles, along with incision, and turning out of the thrombus. Stretching would relieve suffering greatly in these cases, even if incision is objected to.

(2) In external piles and fissures, the external piles being cut off, and the resulting wounds stitched.

(3) In cases of a ring of small venous piles at the anus—often associated with obstruction to portal circulation—in which the stretching removes all discomfort, and attention to bowels, exercise, etc., prevents recurrence. As stated in my former paper, I find sometimes that pinching small piles seems to cure them. I suppose that the veins

thrombose as the result of the injury, and so become obliterated, without pain or risk to the patient.

(4) In well marked cases of internal hæmorrhoids, I operate as I have already described, stretching the sphincter and ligaturing the piles.

I do not mean to advocate ligaturing of piles as against all other methods. But I do mean to say that the addition of stretching the sphincter has removed the operation by ligature out of the category of painful and almost cruel procedures, to the position of being one of the simplest and best methods of treatment.

For a fuller statement of my views, in regard to the treatment of piles, I would refer to my paper in the "Clinical Journal," 26th July, 1893.

CLINICAL NOTES.

(Specially reported for The Clinical Journal. Revised in each case by the Author.)

WITH DR. JOHN PHILLIPS IN THE GYNÆCOLOGICAL DEPARTMENT OF KING'S COLLEGE HOSPITAL.

Dyspareunia.

By this term we mean painful or difficult sexual intercourse. Three of the women whose cases have just been taken have referred to it; it is a delicate matter to touch upon, and every consideration should be shown the patient in our inquiry.

Let us take the *first* case—a woman, aged 48 years, with six children. She tells us that her labours have all been normal, and that the menopause or "the change" (as she calls it) has been completed two years. Until six months ago she enjoyed perfect health. She now complains of intense pain during coition; on further inquiry it is found that the pain is referred to the external orifice, and it occurs at the commencement of the act only.

On examination in the left lateral position, at the moment of inserting the index finger, the patient as you saw gave a start and a cry of pain. On inspection of the vulva and separating the labia externa, we found the usual local signs of the

menopause well developed; in addition, however, there were small patches on the surfaces of the internal labia of a purplish-red colour, and apparently not raised above the surrounding surface; they were most marked near the urethral orifice. On touching them they were found to be intensely tender. We carefully localized this tenderness by means of the blunt end of a uterine sound. Nothing else was found in the pelvis. These tender patches are evidently the cause of her dyspareunia. Nitric Acid was applied on a stick to each patch; this may have to be repeated, and a cure should result; if not, it will be necessary to touch the diseased parts with a Pacquelin's cautery. You will find in the course of practice that this morbid condition is not at all uncommon in women at or about the climacteric. Fortunately, diagnosis and cure are comparatively easy.

The *second* case is an exceptional one. The patient, who is 20 years of age and only married three months, states that she had great loss and suffered severe pain during the first sexual intercourse and since. The pain, however, varies much, sometimes being tearing and cutting, at others stretching in character. The dyspareunia is at the commencement of the act. Her functions are quite regular, and she complains of nothing else.

On examination, which was quite easy and painless, the parts seemed normal to the touch. On inspection, however, the cause of the dyspareunia was evident. Lying transversely across the vaginal orifice you saw a band or bridle of mucous membrane, with attachments on the right and left sides. This condition was produced by a tearing through the base of the hymen, instead of its free edge, during the first sexual approaches, and accounts for the pain and hæmorrhage she told us of. This bridle apparently sometimes acts as an obstacle to intercourse, sometimes not, and the remedy is very simple; she will be taken into the hospital, and the loop removed by means of the Pacquelin's cautery.

The *third* case is unfortunately more common. Referring to our notes, we find that the patient is 30 years of age and has two children, the last seven months ago. Since getting up after the last labour she has had much pain in the left side (especially after walking) and dyspareunia, which latter is gradually increasing in severity; not only is there pain during the act, but a throbbing pain which lasts for some hours afterwards. Three months

ago she had an attack of "inflammation of the bowels," and she is worse since. On examination, we found none of the vulval tenderness observed in the other two cases. The vagina was patulous, and the uterus, though large and in a state of retroversion, fairly mobile and quite painless. In the posterior *cul-de-sac*, and slightly to the left, we felt a small roundish swelling, which was not freely mobile; immediately it was touched the woman called out with pain, and you noticed also that she retched several times. The patient evidently has a prolapsed tender and semi-fixed left ovary, which is the cause of her suffering. A sound was not passed, because it was quite unnecessary as a means of diagnosis, and because of the recent inflammatory attack, which was probably perimetritic in character.

The clinical history of her case is probably this: After her last confinement the heavy, subinvoltuted uterus became retroverted, owing to exertion; the left ovary also became displaced into Douglas's pouch, and remained mobile at first. The attack of "inflammation of the bowels" (perimetritis) resulted in the formation of adhesions which fixed the ovary in its present situation. The results of perimetritis in producing adhesion of an ovary in Douglas's pouch are well seen in this specimen. This condition is a very miserable one for the patient; her ovary is injured at each act of coition, and fresh attacks of perimetritis are likely to occur. Married life at length becomes unbearable; as the ovary lies in close proximity to the rectum, scybalæ will cause great pain as they pass on towards the anus.

The treatment in such a case as this is extremely unsatisfactory. Free saline purgation is the first and very essential step, and we therefore order her the hospital "Mistura Alba" thrice daily until the effect desired is produced. Abstinence from sexual intercourse is very necessary, but although you may order it, it is often quite impossible for a woman in this patient's class of life to follow it out. The only course to be pursued is to admit her into the wards, where she will have physical and sexual rest. Hot vaginal douches will be given, and after a month of recumbent posture she will probably be able to bear a soft ring pessary to support her enlarged and retroverted uterus; temporary benefit will result, but our experience of these cases is that relapse almost invariably occurs, and that no more unsatisfactory class of case comes under our notice.

Amenorrhœa with Infantile Uterus.

This patient has come to know "whether she can marry, as she has never seen anything." She is 32 years of age and looks in robust health; she says that there is nothing the matter with her generally, and that she works ten hours a day as a machinist. Observe that her face is rather of the masculine type, and her voice rather deep-toned and coarse. On further inquiry, nothing whatever can be elicited as to periodic pains or sensations, as if an attempt at menstruation were being made.

On exposing the chest, we find that the breasts are not at all developed, no glandular tissue can be made out, and the nipples are small and as in the male. The thyroid gland is not evident either to sight or touch. We now ascertain the external pelvic measurements by means of the callipers, and find as you see, that they are within the limits of the normal condition.

On making a vaginal examination, instead of the cervix projecting from the vault, we feel a small prominence occupying its position. On bimanual palpation the fundus uteri cannot be detected in either *cul-de-sac*. By exposing the parts with a Sims' speculum, we find that the clitoris is of the normal size, and that the prominence already mentioned has a central orifice, through which a sound can be passed a little over an inch. The uterus is, therefore, extremely small. Rectal examination with the left forefinger enables us to remark the absence of the usual projection of the cervix, and higher up the absence of any ovarian enlargement. To complete the diagnosis, the passage of a sound into the bladder, the finger still being in the rectum, shows us that there is no development of the fundus uteri. We must, therefore, conclude that this woman has an infantile uterus and probably immature ovaries, in fact, a condition exactly represented by this specimen of the pelvic organs, which was taken from a girl of 8 years of age.

As the vagina is of normal length, we can inform the patient that she can marry, but that it is extremely unlikely that she will become pregnant. It is obvious that no treatment can remedy this condition.

You may remember some little while ago, a young girl came here with exactly the same local conditions, but, unfortunately, at what should have been each menstrual epoch, she had severe epileptiform attacks, and she is still under observation: so far treatment has been of no avail.

Subsequent Course of a Case of Total Extirpation for Cancer.

This patient many of you may recollect. She first applied here nearly three years ago, when 31 years of age, suffering from a sharp attack of bleeding. On examination a fungoid mass occupying the posterior lip was found: microscopical examination showed it to be epitheliomatous. The uterus was quite mobile, and the vaginal mucous membrane free. She was admitted, and total extirpation was performed, the broad ligaments being secured by ligatures; the ovaries were also removed. The patient made an uninterrupted recovery. She is a very intelligent woman, and knowing the nature of her ailment, takes every care to follow out all directions which are given her.

She was told to report herself every month for six months after the operation, and then every six months, and to weigh herself every month; these rules she has carried out regularly. She has been through all the symptoms of an induced menopause, which is apparently now completed. We find on referring to her card that her weight has steadily gone up, until eight months ago, since which time it has remained stationary. She has had neither hæmorrhage nor pain.

On examination, we find that the vaginal vault is a *cul-de-sac*, and when you observe it through a Sims' speculum, there is a perfectly healthy stellate glistening cicatrix, instead of the projecting cervix. No bleeding occurs on examination. The pelvis is free from any masses of recurrent growth, and we may fairly conclude that the disease has so far not returned. We propose to keep her under observation another two years (making five since the operation), before deciding to call her "cured."

THERAPEUTICAL NOTES.

Syphilis.—Some of our readers have, doubtless, found cases of syphilis in which Mercury is found to be very ill-borne by the patient, either from idiosyncrasy of the individual or from other reasons; we, therefore, publish the conclusions of Dr. Price (a surgeon in the United States Navy) regarding the use of Copper in this disease, although they are still in an incomplete and tentative condition.

"I consider that the following conclusions may

be formulated as being probably true; although I do not consider that either the number of the cases, or the circumstances attending some of them, allow of positive deductions.

1. Copper exercises a specific action in syphilis, which is especially directed toward the lymphatic system. It is for this reason more radically curative than Mercury.

2. It is slow in removing the skin symptoms of the secondary stage.

3. It prevents the development of mucous patches and throat symptoms.

4. It is a very active drug, and it is wise to omit its use one day in a week, and sometimes more frequently. The signs of its excessive and injurious action are first a voracious appetite, and this is rapidly followed, if the dose is not reduced or the drug temporarily discontinued, by prostration, giddiness, pallor, and a rapid and weak pulse.

5. The average dose of Sulphate of Copper is $\frac{1}{30}$ of a grain thrice daily. It is better to give it with the Sulphate of Iron. It can be given either in pill or solution.

6. This dose is absolutely dangerous in cases of syphilitic cachexia. It produces at once excessive and alarming prostration. If a sufficiently small dose of the drug is given at first, a tolerance of it is gradually established, so that the average dose may, in time, be obtained.

I am inclined to think that in some cachectic cases as small a dose as the $\frac{1}{10000}$ of a grain may be necessary, given once daily. The use of Iron, Arsenic, and Iodide is also usually necessary in old syphilis."—(*New York Med. Rec.*)

Hysteria and Marriage.—The belief is as old as Hippocrates that hysteria is cured by marriage. But Dr. Wythe Cook finds, from his experience, that in most cases of dysmenorrhœa and hysteria among single women, marriage aggravates the disease. Hysteria is by no means cured by marriage, dysmenorrhœa often returns after pregnancy.—(*Amer. Jour. of Obst.*)

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THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 14, 1894.

A CLINICAL LECTURE

ON

TWO CASES OF CHOREA AND A CASE OF HEPATIC ABSCESS.

Delivered at St. Bartholomew's Hospital, Jan. 19, 1894, by

Sir DYCE DUCKWORTH, M.D., LL.D.,

Physician to the Hospital.

I PROPOSE to discuss with you to-day the illnesses of three patients, illustrating two very different morbid conditions, viz., chorea and hepatic abscess. I shall first direct your attention to two cases of chorea that have recently come into my wards.

One of them, that in Elizabeth Ward, a young girl 17 years old, was admitted on the 10th January. Her occupation is that of a waitress in a coffee-house.

The story of her case is as follows: About a year ago she began to be sick directly after her meals, but had no pain after eating. She suffered from frontal headache. The catamenia had been absent for six months. She was treated for these ailments and kept on with her work; but her health was not restored. On the 11th December she suffered from an illness which was in all probability an attack of influenza, and was obliged to keep her bed for a week. She returned to her work on the 6th January—not feeling well, complaining of much headache. She was noticed to be strange in manner, irritable, and ready to cry on trifling provocation. Her memory had also become bad. At the same time she was noticed to be suffering from jerky, irregular movements of the face; and on the day following that on which they were first observed they grew worse, so that she became unable to keep still. She slept very badly at this time.

There is a story of her having brought up blood on the 8th January, whether from the stomach or lungs it is not possible to ascertain. She also said that she spat up streaks of blood. I do not attach much importance to that history. A fortnight ago she declares that her ankles were swollen.

It appears she never had any similar illness before. There had been no history of late of any mental anxiety, strain, trouble, or of over-pressure. There is no history of rheumatic fever or of pains in her joints. On inquiring into her family history it appears that her mother had rheumatic fever, and that her father also had rheumatic fever in his youth; that an elder brother has twice had rheumatic fever; an elder sister has also had it; and another brother had rheumatic fever when he was eight years old. I shall have to call your attention to that rather remarkable family history.

Her aspect on admission was that of a well grown, fairly well nourished young woman. Her expression was noteworthy. I remember calling your attention to the curious *facies* or facial aspect—very difficult to describe, but not difficult to recognize, once your attention is called to it,—the aspect of a person of unsound mind, a silly, unsettled look in the face; a quality of expression, as I have said, very difficult to define, but a physiognomy such as one is quite familiar with in cases of chorea.

An examination of the various systems of the body did not reveal very much.

We found her pulse rather irregular, and of somewhat low tension.

The condition of her heart was examined, and it was found that the apex-beat was in the proper interspace, half an inch within the left nipple-line.

The action of the heart was irregular, as was the pulse, of course; the first sound being somewhat sudden, but no murmur was detected. The area of cardiac dulness was natural.

The chest was well-shaped, and there was nothing wrong noticed in the condition of the lungs.

The abdomen was perfectly natural.

There was a questionable degree of œdema about the ankles.

Her blood condition was rather poor, the patient being slightly anæmic. The mucous membranes were rather pale. Her pupils were both large—a common condition in anæmia.

Her temperature was quite low, being on admission only 96°.

She was put to bed and kept quiet.

We noticed but few, jerky, irregular movements of the limbs, and twitching of the fingers; but still these fidgetty movements, together with the characteristic facial aspect, were of such a character as to leave no doubt about the nature of the case, namely, that it was one of chorea in an early stage.

As I said before, there had been no strain, overpressure, worry, chagrin, or trouble to excite the attack. The only point worthy of note was the occurrence of influenza about a week or ten days before the choreic symptoms began.

The second case—I shall return to the first presently—is that of the little boy in the state bed in John Ward, 12 years of age, who came in on December 30th last. His history is very short. Five weeks before admission this boy had an attack of influenza. Now, when I was preparing this lecture and looking out in Case 1 for an exciting cause of the chorea, as I have just said, I could come upon none. I therefore concluded that the influenza might fairly be blamed for determining, or precipitating this attack of chorea. I had forgotten that when I came to review the notes of this second case; but here again I found the patient had had influenza a few weeks before the chorea had come on. Within a week or so after his attack of influenza, this boy was noticed to have twitching and jerking of the right arm and hand. A day or two later the same thing was noticed in his legs; then the left arm and face began to twitch, and at last he was unable to hold anything in his hands, to feed himself, or to walk.

In this case there was no history of fright or mental strain. The boy was at school and has been in for examinations lately. This is a point I must call attention to, and I will tell you why. It is because the strain of examinations has been noticed in America, where there is great educational pressure, to be connected with outbreaks of chorea. It is found that in the large towns of the United States a great many attacks of chorea occur in June. Now that is just the time of the year when the children are submitted to their annual examination for promotion, and the pressure upon American children at that time is very considerable. They are precocious enough naturally, and very much alive to all that is going on, but when educational pressure is put upon the American nervous, highly-strung temperament, the result is that there is a considerable number of cases of chorea. Examinations have been blamed

in this country, too, and to school pressure, or rather to School Board pressure, since the institution of that system in this country, has been largely attributed the induction of chorea. The "schoolmaster abroad," paid by results, has been responsible for much of the great pressure put upon children, sometimes feeble in body and not too strong in mind, and very often not too well fed; so that they have had in many ways to struggle against nature in the acquisition of knowledge (which, by the way, is a very different thing from being educated). There is no doubt that with the spread of education there has been a spread of chorea. But that is merely a determining cause; it will not produce chorea. The fear of the schoolmaster, frightenings by parents, brothers and sisters may strike great terror into children; but none of these will produce chorea unless the patient has a predisposition to the affection.

These are all determining causes without doubt, The terror of a schoolmaster, the fear of a foolish parent, of a drunken father, of a brutal brother, the teasing of an unkind sister, the "nagging" of an unkind master or mistress are very bad and hard to bear; but they are intolerable to young people predisposed to chorea, and very often determine the disease. Remember, however, there must be a predisposition, and that predisposition, I venture to believe, is a rheumatic proclivity.

We have two conditions, then, in this boy. First, he had influenza and became choreic a week afterwards; second, he had been in for an examination. I leave you to choose which condition determined the chorea in his case. You may think it a little far-fetched to reckon influenza as a predisposing cause; but at this time of day, in this fourth winter of influenzal visitation I am bound to say that the more I see of it the more strange results I find. It really seems as if there were hardly any malady, ailment, or ache that is not attributed, and often with good cause, to influenza. There is no doubt it is a widespread malady, a far-reaching disorder, affecting every system in the body; so much so, that if you get a bad attack of influenza you are certain to be badly smitten somewhere. It may be in the head, the chest, or the abdomen, the alimentary canal, the spinal cord, or the peripheral nerves; there is no limit to the widespread ravages and damage produced by influenza. Of course, during an epidemic of it a great many disorders are called influenza. Nobody can have a bad cold just now

without reckoning himself the victim of it. However, I do consider that influenza when prevalent may conceivably be a determining cause of chorea in predisposed subjects.

Like the girl this boy had a rheumatic element in his family history. His father suffered from rheumatism. The patient had no particular disorder of his heart; he had no murmur, and the area of cardiac dulness was natural. The first sound was, perhaps, a little prolonged at the apex, but he had no physical signs except that.

In the course of a few days a large abscess formed in his scalp, due to irritation of pediculi. This had to be opened.

His movements were exceedingly violent; he had to be tied up; and he could not feed himself.

In a well-developed case of chorea there is no difficulty in making the diagnosis; anybody could tell what is the matter. Great attention should be paid to the state of the heart; murmurs, which occur very frequently in chorea, should be sought for. Many of you may know that there has been much discussion in the schools as to the intimate nature and pathology of chorea. The question cannot yet be considered quite settled. In my own mind I have long had not much doubt about the matter. It has been borne in upon my mind that chorea is one of the rheumatic diseases—a disease that people, especially young children, of rheumatic habit of body, are especially prone to. If you consider chorea as a rheumatism of the brain, I think you will not be far wrong in almost every case. If you ask me, or anyone else, to show you signs of rheumatism at the same time elsewhere, I say it cannot always be done. I am quite content when I see a choreic patient to say, There is rheumatism affecting a certain part of the brain, most probably the cortical area, not affecting the heart or joints in that case, it may be; or one or both of these may also be involved. Or rheumatic joint-troubles may come on after the chorea, while in other cases the chorea may come on after the affection of the joints. That chorea is a rheumatic disease I have no doubt whatever. I do not stay to discuss that point now, because I hope shortly to express these views at the International Medical Congress in Rome.

In the case of this girl, at all events, the rheumatic family history is exceedingly strong. Although there is no personal history of rheumatism, it is as marked in her family as you could wish. She has not had rheumatism in any form

till now, after the attack of influenza, she is smitten with chorea. That is her rheumatism; and she may yet have some joint-affection or some other rheumatic manifestation,—some sore throat or skin-affection due distinctly to the toxine of rheumatism. In the meantime, I am satisfied to believe that the chorea is a rheumatic manifestation in her. Even in her case the heart is a little affected; for the first week after admission it was excited and irritable; and the apex beat is found to be not quite steadily fixed at one point,—at some times it is a little more out, at others a little more in.

One of the peculiar features of this girl's case was that she began "to go off her head." Within forty-eight hours after admission she began to have extraordinary delusions—delusions as to pregnancy, delusions as to pediculi on her head. She was also very restless, wishing to get out of bed. Mental disturbance is sometimes met with, even up to the degree of acute mania. A choreic patient has sometimes become quite insane at the outset of the affection. This disturbance was controlled very well by small doses of Hyoscine, $\frac{1}{16}$ of a grain, subcutaneously injected, being followed by great benefit. She had full doses of Hydrate of Chloral and Bromide of Potassium for two or three days. The delusions having passed off, the chorea developed a little more, but since then has subsided, in response, I think, to specific treatment, namely, treatment by Arsenic, which I consider to be the best remedy for chorea.

In this case, and in the case of the boy, we have employed the same remedy, namely, Arsenic in full doses. By full doses I mean no less than 15 minim doses of Fowler's solution. That is, three times the average full dose as recommended in the books; but there is good reason for increasing the dose very materially in order to be successful in the treatment of chorea. These 15 minim doses are given three times a day for three days consecutively; then their administration is suspended for one or two days to see what the effect is generally. It is a good plan to stop the administration of these large doses for three days, because sometimes you find a patient who, being treated successfully in this way as far as his disease is concerned, otherwise bears it badly; there may be peripheral neuritis, or paralysis of sensation, or paralysis of motion in the limbs, or, again, it may be resented in the usual way, by the

stomach, which becomes irritable, and vomiting is induced.

We have had no trouble, however, in these cases. By suspending the remedy, and then resuming it for a few days more, the effect produced is more marked. We are indebted to Dr. William Murray, of Newcastle-upon-Tyne, for inculcating this method by what may be termed heroic doses of Arsenic. It is a very good plan of treatment. Whatever will cut short the spasms and lessen the duration of the disease is good. You do no harm, and get the patient well some weeks quicker. The average duration of a case of chorea is considerable, indeed, from first to last, ten weeks and a half under all methods of treatment; but I trust that now, having the knowledge that these large doses of Arsenic are efficacious, the time may be reduced. The more violent forms of choreic spasm are best met by Chloral Hydrate in full doses.

I shall not say more on the subject of chorea at this time, but pass to the next case, that of this lad here. He is a young sailor, aged 18. He came in on the 8th December to John Ward with a terrible cough, much wasted, and so obviously very ill that there was nothing for it but to send him to bed at once.

His story is not a very long one, and is to this effect. On the 15th of May last year he went in a sailing ship from Hamburg straight to Maracaybo in Venezuela. He had perfectly good health until the 16th September, when he began to suffer from pain in the right side of the chest on taking breath—a sharp, cutting pain, not continuous. He was short of breath, and his appetite began to fail. The pain got worse, and he became so ill that he had to be taken ashore to the hospital, where he remained for three weeks. There he was repeatedly wet-cupped over his liver. His diet consisted of salt rice and plantains, that is, small bananas. That and wet cupping was all the treatment he got. He sailed for home on the 4th October, still having pain in his right hypochondrium. Three weeks later on the voyage home he began to pass blood with his motions, which occurred ten to fifteen times daily with straining or tenesmus. This lasted for about a week. During the last fortnight of the voyage home he had diarrhoea, headache, and malaise. During this period they seem to have been on short allowance both of food and water. The patient also states that two days after the beginning of his illness, before going into hospital,

he began to spit up bloody stuff, which increased in quantity from week to week. He occasionally vomited some bloody material. He had certainly wasted materially. Up to the time of this illness he had always been healthy and strong and of temperate habits.

When he came in he had severe cough and very copious expectoration of pinkish stuff. On examination this was found to contain bile; but no very distinctly formed cells could be recognized as belonging to the liver. His temperature was 102° on admission. There was no jaundice. The tongue was dry, with a thin white dorsal fur.

On examination of the chest the right side was prominent, the intercostal spaces full. The right side moved hardly at all. The vocal vibration was absent from the front of the chest below the second rib; and dulness extended from the liver dulness right up to the second rib. The breath-sounds were absent all over the dull area, as also were the voice-sounds. Above the dull area the breathing was puerile. On the left side there was exaggerated breathing, the left lung doing the greater part of the respiratory work. Behind, the percussion was good on both sides. Thus, you see, there were curious physical signs—a large dull area in front, and all perfectly normal behind.

The heart's apex beat was displaced about an inch outside the left nipple-line. The sounds were natural.

Abdomen—The dulness of the liver did not extend below the edge of the ribs, and was not felt to come down on full inspiration. The patient had to sit up, being unable to lie down.

With these facts before us, and with these physical signs we had to make a diagnosis. It was not a very difficult one. The history and physical signs all pointed to the great likelihood of there being an abscess of the liver, especially in the right lobe, in the dome-shaped part which underlies the diaphragm. More than that, the indications were that this abscess had ruptured the diaphragm, had caused adhesions to occur at the base of the right lung, had passed into the lung and bronchi, by which channel it was liberating itself, so that its contents were spat up by the patient.

That was the diagnosis; and that, of course, indicated only one method of treatment. Mr. Langton put an exploring needle into the fifth intercostal space in the nipple-line, and drew out a quantity of reddish matter, exactly similar to that which the patient was spitting up. He then

made a free incision over the fifth rib, of which he removed from $1\frac{1}{2}$ to 2 inches, and got into a space which he supposed was part of the thoracic cavity, inasmuch as on putting his finger down he felt the floor of the diaphragm, and found a quantity of thick curdy pus mixed with blood. There was about half a pint in the cavity. No communication could be felt through the diaphragm, adhesions having taken place at some point; and so a large incision was made through the pleura directly into the abscess. Immediately after the operation the patient's temperature fell. For the few days previously while we had him under observation, it ranged from $100-101.4^{\circ}$; but it fell immediately after the operation to subnormal, and never rose again. There was complete relief; he was able to lie down; he spat up hardly anything more, the abscess having been freely opened, and the contents let out that way. The patient began to recover his appetite and put on flesh. On admission he weighed 6 st. 9 lb.; when last weighed he registered 8 st. 1 lb. The patient is now quite well and is going to Swanley to-day. There is a perfectly sound wound. (The patient was exhibited.)

We shall now consider a few points about hepatic abscesses generally.

There are two great divisions of hepatic abscesses. There is the TROPICAL ABSCESS, as seen in this case. But hepatic abscess is very rare in the Brazils and the West Indies. It is an East Indian malady, a disease of the East Indies, China and the Straits Settlements. This tropical abscess is generally single, and often very large.

The second form of hepatic abscess may occur anywhere, and is known as a form of pyæmia. PYÆMIC ABSCESSES are generally small and multiple.

Much discussion has raged over this subject amongst East Indian doctors and professors of pathology as to the causation of tropical abscess of the liver; and the matter cannot yet be considered settled. The question has been urged whether hepatic abscess is not always due to dysentery. Those who hold that view say that the patient first acquires dysentery and subsequently has abscess, the one being the result of the other, that products are taken up from the large intestine, which is the seat of dysentery, by the portal vein and lodged in the liver, where, by a process of embolism, they set up an abscess. If that were the case one would expect that the

abscesses there would be multiple rather than single. There is, however, no doubt of the great frequency of hepatic abscess as a sequel to dysentery. On the other hand, tropical abscess occurs in people who have never had dysentery at all, so that dysentery cannot be blamed in these cases. In other cases dysentery follows hepatic abscess. The probability is, I would say, the certainty is, that tropical abscess of the liver may occur primarily in some cases; and that thus there are cases in which suppurative hepatitis,—inflammation running into a purulent suppurative stage,—is the result of climatic conditions. It used to be thought that hepatic abscess was due simply to great solar heat. No doubt the disease does occur in hot climates, and not in temperate ones; and heat may have something to do with it, but not directly. I have no doubt the question will be settled before long by the discovery of some specific organism, some microbe, which thrives in certain tropical districts, and which is taken into the system—probably swallowed with either the water or the food—and so reaches the alimentary canal, and, lodging in the liver, there sets up abscess. It has been thought that the same poison that causes hepatic abscess also causes dysentery, both being common results of the same poison. It may be so; but if we find one case where there is a distinct history of the absence of dysentery and the presence of a large abscess in the liver, some other cause of its production must be looked for.

The symptoms, of course, vary very greatly according to the size of the abscess and the part of the liver in which it occurs. Sometimes it is a very obscure disease. Indeed, it may not be recognized until it has burst into the colon, the stomach, the pleura, or the pericardium, in the latter case with an immediately fatal result. Such cases were more common in former years than now. People do not now stay so long in the tropics as they used to do. They come home more frequently, get into better health, and so resist both hepatic abscess and dysentery more successfully.

These abscesses generally occur in young people. It is nearly always a disease of men. Curiously enough, European women living in countries in which hepatic abscess is common are very seldom affected.

Again, people with tubercular predisposition, members of families predisposed to phthisis, and

sent to the East to avoid it, are, more than others, prone to suffer from abscess of the liver.

Once an abscess is suspected it should be looked for by probing with needles. Immediately it begins to point it should be treated lest it burst into some situation where a dangerous or fatal result would ensue. As I said, cases are now less frequently met with than formerly. No doubt with increased sanitation in the different parts of our Eastern empire hepatic abscess may be expected to become less frequent; and as Europeans stay less time in tropical countries than they used to do they may be less exposed than our forefathers were.

The colour of the pus in a hepatic abscess varies. It is sometimes like ordinary pus; but my experience is that it is generally red. The quantity of discharge may be enormous.

Such cases may not do as well as this one has done. They may go on for months. Together with Mr. Langton—who has operated on three cases for me—I saw a case in private, that of an officer who was attacked with the disease in India, from whom a most marvellous amount of discharge came away for months and months. It seemed as if his liver were melting away, until I began to imagine he could not possibly have an ounce of liver left. Where it all came from I hardly know. That gentleman is still alive and very fairly well. Of course he will always be an invalid. We must suppose he has now a very inadequate liver.

The great improvement in practice has come from modern surgery with all its cleanly appliances. Bold incisions, free drainage, and perfect cleanliness—that is the secret of success in the treatment of hepatic abscesses; and many cases lost in former years would in these days be saved without any difficulty by the application of modern surgery. I sometimes tell my surgical colleagues that surgery has now become a very important part of the practice of medicine.

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18, Middle Street, Aldersgate, London, E.C.

A CLINICAL LECTURE ON PROSTATIC DISEASE, With Special Reference to its Diagnosis and Treatment.

Delivered at the Yorkshire College, January, 1894, by

A. W. MAYO ROBSON, F.R.C.S.,

Hon. Surgeon to the General Infirmary at Leeds; Professor of Surgery in the Victoria University.

GENTLEMEN,—Whatever may be the cause, we must accept the fact that at any time past middle age the prostate is liable to a pathological enlargement, which may lead to an obstruction in the passage of urine, to the formation of a pouch in the bladder, and to other complications which we will shortly mention in detail.

The theory that enlargement of the prostate is a peculiar degeneration of old age, it seems to me, does not meet the case, as the affection may occur in middle life, is not always associated with signs of degeneration in the vessels or elsewhere, and frequently is not present when other degenerative changes are well marked.

Nor can it be explained satisfactorily on the hypothesis that the enlargement is only a secondary change, dependent on a primary depression of the floor of the bladder itself.

The theory which, homologically, physiologically, and pathologically seems to accord with what we know of prostatic disease, is that the prostate is essentially a part of the generative system, and like its homologue, the uterus, is liable to certain forms of new growth resembling fibro-myoma, only containing more adenoid tissue; and just as fibromyomata of the uterus tend to occur more frequently during the latter half of sexual life, so in the male who is sexually potent to old age, the development of prostatic enlargement might be expected, and actually does occur, any time after middle age.

There are at present under my care in the infirmary six patients suffering from symptoms dependent on enlarged prostate, and if you will avail yourselves of the opportunities of observation thus afforded, you will find that five are advanced in years, but that the sixth is only 48. They have all been troubled with frequent micturition, more marked at night than during the day, until in one

of the most marked cases the patient has had to get up every hour, and has consequently suffered severely from want of rest. This symptom depends at first on a congested condition of the neck of the bladder and prostatic urethra, and later is intensified by the retention of so-called residual urine and the irritation of this when it becomes ammoniacal.

Besides *nocturnal incontinence*, there are other subjective symptoms, as a *difficulty in starting* the act of micturition and an *interruption of the stream* from time to time, caused by spasm of the sphincter vesicæ, or in the latter case by a mechanical block due to an enlarged middle lobe.

You will find in all the cases that there has been a marked *feebleness* of the stream, either due to the mechanical obstruction or to atony; there may be elicited certain sensory disturbances, as a feeling of *urgency* on micturition and of *discomfort after*, and in some a *dull aching* in the perineum and rectum, often extending to the back and to the inner surfaces of the thighs; or in the more marked cases, a feeling as if the *rectum were plugged*, giving rise to some tenesmus. In the more advanced cases *complete retention* is apt to occur, dependent on an aggravation of the urethral congestion or on inflammatory swelling at the neck of the bladder; or there may be *incontinence*, due to retention with overflow, this coming on as soon as the intra-vesical tension is sufficient to overcome the resistance of the sphincter of the bladder; and with these symptoms the urine is apt to assume the changes characteristic of *cystitis*.

It is not to be wondered at that in the more advanced cases there should be a general *failure of health* with loss of flesh.

Occasional attacks of *fever* with digestive disturbance, dry tongue, and slight albuminuria, at times ending in uræmia, are apt to occur. Later, a low form of septicæmia may end in death from exhaustion, or kidney disease may lead to a fatal termination from uræmia.

Besides the symptoms which may be elicited by questioning the patient, the surgeon can gain much further information by digital and instrumental manipulation. With a patient on his back and the knees drawn up, the surgeon's well-lubricated right index finger passed into the rectum can gain much information as to the size and consistency of the lateral lobes of the prostate; and when the bladder is empty and the warm left hand is placed at the same time over the pubes, the upper surface

of the prostate may often be felt and the middle lobe mapped out. I would advise you to practise this bimanual method of examination, which is of marked utility in vesical diagnosis, and in no disease more useful than in the one we are now considering.

It is next of importance to ascertain the amount of residual urine, and this is done by passing a catheter, after the patient has emptied his bladder as far as he is able by voluntary effort.

You will notice that all the patients at present under observation have this symptom, more or less marked, one patient having only 2 oz., another as much as 23 oz. of residual urine.

In using a catheter you must be most careful to secure your instrument being aseptic; in the case of a silver one, by boiling, or of a soft catheter, by dipping it in boiling water, and afterwards leaving it for a little time in a 1 in 1000 Perchloride of Mercury solution. Not less important is it that you should avoid all violence, remembering that gentle manipulations will accomplish more than force, and that the latter is dangerous in the extreme, and will intensify all previous symptoms, and at times convert a simple case into a serious one.

Besides the amount of residual urine, the passage of a catheter will tell you where the obstruction is, for instance, if a firm instrument is arrested at a point seven inches or more from the meatus, you know that the prostate produces obstruction, and if a steel instrument or a straight flexible catheter is arrested, and a coudée catheter will pass, you know that the middle lobe is enlarged.

If instruments readily pass, but the urinary outflow is embarrassed, you may reasonably infer that the middle lobe is enlarged, and if the catheter shows the urethral canal to be lengthened, it will give you some idea of the antero-posterior diameter of the prostate.

When a hollow instrument has entered the bladder, the force of the stream of urine through it will give you some knowledge of the tonicity of the bladder. If the finger be passed into the rectum while a firm catheter is in the bladder, the amount of tissue between the catheter and the digit can be easily estimated, thus giving a fairly accurate idea of the size of the middle lobe of the prostate.

As a rule, the use of the cystoscope is not necessary in these cases; but if employed, it will furnish evidence not only of the condition of the

interior of the bladder, and of the presence or absence of calculi incapable of being felt by the sound, but also of the state of the ureters and kidneys.

Treatment.—Having made the diagnosis, the next question to consider is that of treatment, and I need scarcely say that if you happen to discover prostatic enlargement which is producing no symptoms, and in which there is no residual urine, you will not be justified in interfering in any way lest you expedite trouble which may be deferred indefinitely.

If, however, the enlarged prostate is producing symptoms, and there is a certain amount of residual urine and vesical irritability, what can we do?

We must attend to the general health, and if required give some tonic, for instance, a pill containing a grain of Dried Sulphate of Iron and $\frac{1}{2}$ grain of Extract of Nux Vomica is useful; and if catheterism is required it is desirable to administer an antiseptic which will be discharged as such in the urine, and for this Boracic Acid or Salol will be found useful. As for any medicine to exercise a specific effect on an enlarged prostate, I believe all the reputed remedies are futile, and not worth troubling with.

We should give a fair trial to palliative treatment in the shape of catheterism. If the residual urine is only small in amount, say two ounces, the regular passage of a catheter at bedtime is all that may be called for, if more than that amount of urine is retained, say five ounces, catheterism night and morning will probably be required, but when the amount of residual urine is greater than this, and when the catheter has to be used frequently, the question of operative treatment will have to be considered.

Whenever you can relieve by milder means, avail yourselves of them, and do not resort to operative treatment until the milder measures have had a fair trial.

We have a peculiar interest in Leeds in the treatment of prostatic affections, since it was in this theatre that my late most distinguished and most esteemed colleague and friend, McGill, initiated the operative treatment of the disease, as previous to the publication of his series of cases, surgeons were content either with palliative treatment or with quite inadequate methods now little practised. Among these minor operative procedures which I must mention to you, but at the

same time would not advise you, as a rule, to rely on, are:

(1) Overstretching of the prostatic urethra through a perineal incision.

(2) Interstitial injection of Iodine, Ergotin, Carbolic Acid, Sclerotic Acid, etc.

(3) The application of the continuous current of electricity by means of the N. pole inserted into the prostate through the rectum, while the P. pole is applied to the abdomen.

(4) The application of the galvano-cautery to the prostatic urethra by means of instantaneous flashes.

(5) Division of the bar at the neck of bladder by means of a cutting instrument inserted through the penis.

(6) Division of the obstruction by means of the electro-cautery inserted through the penis.

The only operations worth considering in detail are:

(1) Perineal prostatotomy or prostatectomy with drainage.

(2) Supra-pubic prostatectomy.

(1) The perineal operation is practically median urethrotomy, followed by exploration of the prostatic urethra by means of the finger introduced through the wound, and then by incision of the gland or by excision of as much of the obstruction as can be effected through the opening in the perineum, after which a drainage tube is inserted into the bladder. The projecting portion may be crushed by the finger, or by forceps, or torn away by instruments, or excised by means of the prostatectome; or the channel may be enlarged by means of Paquelin's cautery or the galvano-cautery. Only an enlarged middle lobe can be dealt with in this way, and that not always effectually, as in many cases it is quite impossible by this route to reach, much more to remove, the projecting portion of the prostate. Although this method may occasionally give a brilliant result, it is generally inefficient, and it must not be forgotten that the mortality, calculated from a considerable number of cases, is 13.6 per cent.

(2) We come next to consider the operation of supra-pubic prostatectomy, which you have seen performed by me or one or other of my surgical colleagues on several occasions. The last case on which I performed the operation, was on a man, aged 62, who had led a catheter life for six months, and who is at present in the end bed on the right in No. 3 ward; and although the operation

was only performed a week ago, the patient is sitting up, and is beginning to pass a little urine through the penis, though, of course, the greater part still comes through the healing supra-pubic incision. The wound is healthy, and the urine, though offensive on admission, is now quite aseptic, as the patient is taking the Boracic powder previously mentioned, and in consequence the sore surface is constantly bathed with a mild aseptic if not antiseptic secretion; but in order to avoid the possibility of septicity, we have the bladder gently syringed daily, by inserting the nozzle of a syringe into the penis, and washing the urethra and bladder through with warm Boracic lotion.

In performing the operation, you noticed that the skin of the hypogastrium had been aseptitized by the application of a carbolic dressing for twenty-four hours before the case was brought to the theatre.

After a full-sized catheter had been inserted, and the bladder thoroughly washed out, chloroform was administered as being the safest anæsthetic in old subjects, especially in those liable to chest affections, which, in fact, is often the case in senile hospital patients. The rectal bag was then introduced and filled with six ounces of water, after which the bladder was filled with Boracic lotion through the catheter previously introduced to wash out the bladder.

The quantity of water in the rectal bag should not exceed 6 to 7 oz., as a greater quantity has been known to produce serious symptoms by overstretching the rectum.

The amount of lotion required in the bladder is variable, and may usually be estimated by placing the hand over the hypogastric region while the injection is being made, for when the bladder is sufficiently filled it can be readily felt as a rounded swelling above the pubes.

The peritoneum is usually lifted by the rising bladder, but in one case I found it adherent to the pubes as the result of a former operation, and I had to open it deliberately to reach the fundus of the bladder, from which I stripped it, afterwards closing the peritoneal wound before making the vesical opening.

Having made an opening into the bladder sufficiently large to admit the right index finger, the urethral orifice is at once felt for and the nature of the obstruction explored. If the middle lobe be enlarged it may at once be snipped off by the ring-shaped forceps or by McGill's scissors.

If there be a ring of hypertrophied prostate around the internal urethral orifice a V-shaped piece may be taken out of the floor of the ring, and this is best performed by means of the scissors mentioned, a cut being made first on one side and then on the other, the intermediate portion being removed by the ring or by other forceps. If the lateral lobes are much enlarged and are obstructing the passage, the index finger can easily be insinuated within the capsule through the incisions already made and masses can usually be enucleated, like small fibroids from a myomatous uterus.

All loose shreds of tissue should be cut off, and the bladder is then washed out with Boracic lotion at a temperature of 105° to 110° F., in order to cleanse the interior, to wash out clots, and to arrest hæmorrhage, which I have never found excessive.

Should the bleeding prove troublesome, a solution of Hazeline might be employed as an injection, or, if necessary, the wound might be packed around the tube with Iodoform gauze. A full-sized drainage tube is inserted, and the wound is closed by means of two or three silkworm gut sutures, which must take up skin and aponeurosis on either side.

A piece of double Cyanide gauze is laid over the wound, and over this a dressing of wood wool in a gauze bag, and this is changed from time to time by the nurse.

The patient is always got out of bed as soon as possible, often on the third or fourth day.

The disease being so common, you would naturally think the operation would also be very common, but this is not so; for instance, I have only performed eleven prostatectomies, though I have seen or assisted in a great many more. Why is this?

In the first place, the greater number of cases can be relieved by less heroic measures, and, in the second place, many cases come under our care at a time when kidney and other organic disease has rendered them unfit to bear a major operation.

The indications which I usually consider to call for operation are as follows:—

Distress due to obstructed outflow of urine from the bladder, depending on enlarged prostate which cannot be sufficiently relieved by catheterism or general treatment, the patient being otherwise capable of enjoying life and being free from

serious kidney affection or other disease which would render any operation dangerous.

With these provisos, I think, we may pronounce supra-pubic prostatectomy to be a less dangerous operation than is usually thought, and one which certainly should have a smaller mortality than that mentioned in Mr. Mansell Moullin's tables, where it is given as 20 per cent. I have thus far not had to record a death, but this is probably quite as much due to my good fortune in having had suitable cases as to attention to details at the time of operation and afterwards. I know that six out of the series of eleven cases are living and well now, three or four years after operation.

In conclusion, Gentlemen, let me advise you to give every attention to the beginning of these troubles in cases that may come under your care in the future, to be especially careful with regard to the first catheterism and to bear in mind that when other means have failed, if they do, you may be able to hold out to your patient a fair prospect of considerable relief by operation.

ORIGINAL ARTICLE.

THE ETIOLOGY OF THE DISEASES PECULIAR TO WOMEN.*

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ALTHOUGH morbid conditions may develop in connection with the genital system of the woman at any time of her existence intervening between "the cradle and the grave," it is beyond question true that there are in her life four great climacteric periods, in connection with which the great bulk of the diseased states to which she is liable develop; in which I should venture, at a rough estimate, to assume that nearly eight-tenths of all these conditions take their rise. These climacterics may thus be presented at a glance:

1st. Puberty, marked by progressive metamorphosis of the ovaries and resulting ovulation and menstruation.

2nd. Marriage.

3rd. Child-bearing.

4th. The menopause, or change of life, marked by retrograde metamorphosis of the ovaries and resulting cessation of ovulation and menstruation.

FIRST CLIMACTERIC.—It is the ovaries which mark, by their rise to and fall from power, the beginning and the end of woman's functional career. Hippocrates, over two thousand years ago, enunciated the dictum, *propter uterum mulier est*. It was left for our own Peaslee to correct him by changing his formula to *propter ovaria mulier est*, which is the motto which he places at the opening page of his work upon ovarian diseases.

True it is that with ovarian advance and retrogression a simultaneous change occurs in the uterus, but no one will to-day dispute the fact that the first constitutes the cause and the second the result; that the change in the first is the phenomenon, and that in the second the epi-phenomenon.

From the birth of a female child to its thirteenth year, the girl is being prepared by nature for meeting the first great climacteric of her life; and fortunate is it for her if surrounding circumstances do not interfere with her development, or the ignorance or neglect of her guardians defeat the accomplishment of the result. During the first two-thirds of this time of preparation the uterus, ovaries, and adnexæ of the child remain almost entirely dormant. Then, as she advances to the third period, they begin to undergo development, and toward the end of this period this development becomes accentuated, rapid, vigorous, and perfect. Then the ovules break away from the Graafian follicles, the Fallopian tubes seize them, and pass them onward to the engorged and swollen uterus, and the great issue which translates the child from girlhood to womanhood has been met.

If the changes preparatory to this event have been properly performed, all goes well; but if from any cause the growing uterus has been checked in its development; been made misshapen so as to resemble a gourd, or been developed fully for half its bulk and made atrophic in the other half; or if the ovaries at the time of call to duty are wholly or in part undeveloped and incapable of performing their functions; or if the Fallopian tubes or vagina are constricted or impervious, a variety of so-called diseases show themselves, all of which are difficult of cure, and many of which are entirely incurable. If mal-

* Read before the New York County Medical Association.

formed ovaries exist, the most agonizing ovarian dysmenorrhœa, amenorrhœa, or emansio-mensium, with their sometimes co-existing hysteria, hysterio-epilepsy, and even epilepsy, show themselves and hold out to the poor woman the prospect of a life of invalidism, or at least a lengthy and discouraging course of treatment. Who in this room does not recall many cases of this kind, which, beginning with the very inception of menstrual life, have proved most rebellious to curative treatment? If the Fallopian tubes have been strictured by want of development, atrophy, or constriction by outlying lymph, the girl suffers from severe pain at menstruation, or later in life becomes an invalid on account of sterility or tubal pregnancy. If the vagina is constricted or impervious, retention of blood in it or in the uterus becomes an important factor for evil.

Suddenly, the occurrence of this climacteric, created solely by ovarian dictation, makes an imperious call upon organs hitherto insignificant, and finding them unequal to the demand, a long list of ailments develop themselves as a consequence. On the first day of a month, the girl has been remarked upon as a type of female health, vigour, and beauty; on the fifteenth of that same month, the imperfect way in which she has grappled with the functions of her first climacteric have assured her physician that years of invalidism in all probability lie before her.

By the patient's friends, even by her physician, if he be a short-sighted and superficial observer, the trouble which has occurred is attributed to cold, to fatigue, to mental anxiety, or the like; but "the inexorable logic of events" is destined to belie this hope. If the chief symptom be dysmenorrhœa, the condition is carefully classified into "neuralgic, ovarian, congestive, or obstructive," and the physician very properly strives to overcome it by using his best efforts to meet a disability, which is commonly due to imperfect performance of those changes which mark the period styled "puberty." If the vagina be constricted or the cervical canal be impervious, they are distended and kept open; if the ovaries are at fault, general treatment by nerve and blood tonics, exercise, and calisthenics, and local treatment by pelvic massage, and electricity and galvanism, are appealed to, and after a variety of means have been tried in vain, and grave symptoms still exist, the process of ovulation is abolished, and the evils dependent upon it relieved by the humane, scien-

tific, and entirely defensible operation of removal of the tubes and ovaries.

Fortunate is it that many evils entailed upon the woman by "puberty" are sometimes susceptible of complete relief. There are, however, some which project their influence into her future life and show themselves after marriage in various ways. For example, a deformed uterus may be the cause of frequent abortions, which no resource of art is capable of preventing; constricted tubes may give rise to hæmato-salpinx and peri-metritis, the outcome of peri-salpingitis; while obstruction to the transit of the fructified ovum into the uterus commonly gives rise to ectopic gestation.

Surely it is no exaggeration to style this one of woman's great climacterics, one of the periods in her existence which is fraught with the profoundest import for her health, her usefulness, and her happiness. If it be true, as I have said, that the period intervening between a girl's birth and her passage into womanhood is the time of preparation for this, her first great climacteric or critical period, much as to her fitness for meeting the issue will depend upon the proper physical development accomplished in this preparatory time, and all those who watch her career—physician, mother, and teacher—should join hands in sending her into the arena as thoroughly prepared as it is possible to make her for her new functions.

SECOND CLIMACTERIC.—The first climacteric being passed, the girl emerges from it a young woman, and enters into that period of her life which intervenes between puberty and marriage—speaking approximately, a period extending from the thirteenth to the twentieth year, covering about seven years of her existence. Although during this time falls, muscular efforts, exposure to cold, and pathological changes of tissue may, of course, produce disease, this is, to one who has successfully met the issue of puberty, usually a cycle of health and happiness. Then occurs a second climacteric, which sometimes proves a most baneful one to the healthy and robust woman who enters into it—marriage.

There are three morbid states which now frequently show themselves; first, the development of vaginismus, or spasm at the vaginal outlet; second, specific vaginitis, endometritis, and salpingitis; and third, the manifold evils resulting from the induction of abortion by women who resort to this fatal and criminal practice, not to protect their characters from disgrace, but merely to suit their social con-

venience, or from dread of the dangers of child-bearing.

Of the first and third of these I shall say no more, as the management of the first is quite simple and generally understood; and as a mere mention of the third is all that I deem necessary. Of the second I shall speak more fully, and venture to quote upon it from an address given by me which has already appeared in print.

Until the last twenty years specific urethritis was regarded, in the male, as an affection of the most trivial import, as rapidly passing off, leaving few serious sequelæ, and offering itself as an excellent subject for jest and good-natured badinage. About two decades ago Dr. Emil Noeggerath published a dissertation upon this affection, which will for ever preserve his name in the list of those who have accomplished good for mankind, and give him claim to the title of benefactor of his race. This observer declared, (1) that out of growing young men, a very large portion prior to marriage have specific urethritis; (2) that this affection very generally causes urethral stricture, behind which a "latent," or low grade urethritis is for many years prolonged; (3) that even as late as a decade after the original disease has apparently passed away, the man may transmit it to a wife whom he takes to himself at that time; and (4) that the disorder affects, under these circumstances, the ostium vaginæ and urethra, and thence passes up the vagina into the uterus, through the Fallopian tubes, where it creates specific catarrh, and by this disease produces oöphoritis and peritonitis, which become chronic, and often end in invalidism, and sometimes even in death. For this essay Dr. Noeggerath was assailed by ridicule and by contradiction. The matter has now been weighed in the balance, and admitted to its place among the valuable facts of medicine.

Let me tell you my estimate of specific urethritis as a factor in the diseases of women, and let me assure you that I take no peculiar or exaggerated views concerning the matter. What I say will be vouched for by all progressive practitioners of gynæcology to-day. Specific vaginitis, transmitted to virtuous women by men who are utterly ignorant of the fact that the sins of their youthful days are at this late period bringing them to judgment, is one of the most frequent, most active, and most direful of all the causes of serious pelvic trouble in women—one which meets the gynæcologist at every turn, and one which commonly proves in-

curable except by the dangerous procedure of coeliotomy.

Think for a moment of the terrible position in which a high-minded, upright, and pure man finds himself placed without any very grave or unpardonable fault on his part. At the age of 19 or 20, while at college, excited by stimulants, urged on by the example of gay companions, and brought under the influence of that fatal trio lauded by the German poet—"Wein, Weib, und Gesang"—the poor lad unthinkingly crosses the Rubicon of virtue! That is all! On the morrow he may put up the prayer, "Oh, give me back yesterday!" But yesterday, with its deeds and its history, is as far beyond our reach as a century ago, and returns at no man's prayer!

Four or five years afterwards, this youth goes to the marriage-bed, suffering, unknowingly, from a low grade of very slight latent urethritis, the sorrowful memento of that fatal night, which has existed behind an old stricture, and a result is effected for the avoidance of which he would most gladly have given all his earthly possessions.

All this sounds like poetry, not prose; like romance, not cold reality. But there is not a physician in this room who does not know, and who will not at once admit, that every word that I have uttered is beyond all question true, and even free from exaggeration.

I mentioned, in speaking of the grave duties demanded by puberty, that one of the most important functions of the physician in regard to the development of the girl during the thirteen years which precede it, is to instruct her and her guardians how to prepare her for the approaching issue. In language no less strong I would here insist upon the physician's duty to instruct men in all stations of life as to the importance of a "clean bill of health" in reference to gonorrhœa, both acute and chronic, before the marriage contract be entered upon.

Until a very late period the plan universally followed has been this: The man about to be married went to his physician, told him the history of a gonorrhœa, and asked if, now that all discharge appeared to have ceased, any danger would attend his consummating the tie. The physician would ask a few questions, examine the virile organ carefully as to discharge, and if the "outside of the platter" appeared clean, give his consent to the union. The evil which has resulted from this superficial and perfunctory course has been as

great as it has been widespread. To-day the question of stricture, a slight, perceptible "latent gonorrhœa," with its characteristic "gonococcus," is looked into, and not until all trace of disease is eradicated is permission given for the union. A marital quarantine is as necessary to-day in social life as a national quarantine is for contagious diseases in general.

Few men, however eager for matrimony they may be, would run the great risks attendant upon precipitancy if they only knew of them clearly and positively. In no field of medicine is the old adage, "Prevention is better than cure," more important than in this one. If physicians would do their duty fully in this matter, how many unfortunate women now languishing from "pyosalpinx" would in the next generation be saved!

THIRD CLIMACTERIC.—Let us suppose that the girl has passed through puberty and become a wife, and that no evil has thus far befallen her in either of these stadia of her course through life. The probabilities are that within a year or two she will be called upon to face still another climacteric—that of utero gestation and child-bearing.

The passage of the human foetus through the short, mucous-lined canal which leads from the superior through the inferior strait of the pelvis is attended, even in labours apparently normal, by certain traumatisms which have a most disproportionate bearing upon the health of the woman who suffers from them. The reasons for this are not far to seek. Slit the cervix uteri or the perineum freely with a bistoury in a non-pregnant woman, and little, if any, serious injury would result. The parts would cicatrize, and nature would prove herself abundantly able to deal with the trouble. But let a laceration of these parts be effected by the child's presenting part during labour, and a series of events takes place which casts its baleful shadow over the future life of the woman. This unquestionable fact is due to two circumstances which render the parturient woman peculiarly vulnerable to traumatism exerted upon the genital tract in any part of its extent. These circumstances are the following: 1. From the moment of fixation of the ovum upon the uterine wall until the end of gestation a rapid hypertrophy of all the sexual organs occurs. The only phenomenon in the physiology of the pregnant woman which is as wonderful as this is that retrograde process of involution which restores these parts to

their normal state, so that at the end of eight weeks the immense uterus, the voluminous broad ligaments, the copious vagina, and the distensible perineum return to their lessened state of non-puerperality. 2. Wounds made in the genital tract of a non-puerperal woman are bathed during their repair by a bland mucus which is secreted by the glands of the cervical canal and by the vaginal membrane. Similar injuries inflicted upon the parts of a parturient woman are bathed by the lochial discharge, made up of ichorous material, holding in admixture flaking decidua vera, disintegrating placenta, and decomposing blood, which, even under favourable circumstances, set up a sapræmia, and, under unfavourable ones, tend to the development of a dangerous septicæmia.

Such wounds are not prone to heal by nature's efforts, often assume bad features, and interfere greatly with that wonderful and subtle process of involution just alluded to. As a consequence an arrest of retrograde metamorphosis often affects vagina, uterus, uterine endometrium, and broad ligaments, and creates a marked disproportion in the normal relations of all the pelvic viscera at the same time that it weakens those supports which formerly sustained them. In this way we often see as direct and remote consequences of laceration of perineum and cervix uteri some or all of this long list of pathological results:

1. The vagina suffering from subinvolution and unsupported by the perineum, begins to undergo prolapse, its posterior and then its anterior wall pressing downward into the vulva.

2. Prolapse of one wall soon deflects a wall of the rectum, and that of the other of the bladder, and misdirection of the forces of these viscera add their influence to increasing vaginal prolapse.

3. Traction by vagina, rectum, and bladder soon affects the uterus, and descent of this organ begins to show itself.

4. The uterus is the more prone to descend under traction because involution has been rendered imperfect in it, and its increase of weight and feeble support from subinvolved broad and round ligaments render it helpless.

5. Menstruation now occurs, and (a granular, almost fungoid, condition of the endometrium, existing in consequence of impaired involution,) menorrhagia supervenes and adds its depreciating influence to the concatenation of morbid influences which are rendering the condition of the poor woman more and more wretched.

Could such a collection of untoward symptoms be possible from wounds inflicted by bistoury or scissors upon the perineum and cervix of a non-parturient woman? Not at all. Such results can be accounted for only by the interference of these traumatisms with the performance of an important physiological function, upon the fulfilment of which depends a return of every puerperal woman to the domain of health.

To sum up this part of my subject, there can be no doubt of the fact that a large proportion of the diseases peculiar to women are due to injuries inflicted upon the genital tract during labour, and that among them may be numbered, subinvolution of uterus, vagina, and perineum; fungoid endometritis; uterine catarrh; menorrhagia and metrorrhagia; any of the displacements of the uterus; prolapse of bladder, rectum, and vagina; cystitis; and secondary derangement of the blood and the nervous system, which react upon all the organs of the body.

To overcome these pathological results the obstetrician to-day repairs lacerations of cervix and perineum, lessens the volume of uterus and vagina, shortens the round ligaments, and scrapes away the entire lining membrane of the womb. When it becomes the accepted and universal practice to examine for these traumatisms after every labour, and to cure them by surgical means as soon as practicable after delivery, the diminution which will occur in the annual crop of sick women will be very striking.

Some years ago a writer upon this subject in an American periodical ventured to hint that avarice on the part of medical men greatly increased the sum total of gynecological operations. I am convinced that he was in error. A desire to multiply the gynecological work of the world ought to prompt an avoidance of the only means of cure possible in such cases as those of which I speak, and dictate the policy of leaving them to the baleful influences which have been mentioned. But the policy of the profession of medicine, which dates back through the ages as one always pledged to honest striving after the real good of society, needs no vindication at any man's hands to-day!

Some years ago, in my clinic in the College of Physicians and Surgeons of this city, being desirous of impressing the facts which engage our attention to-night upon the students, I requested them to notice how frequently each patient who

appeared before them would refer the cause of her disease to one of the critical periods mentioned, and agreed that we would review the subject at the end of a month. The result served to fix the fact most clearly in their minds by the frequent, indeed, almost constant, recurrence of the phrases, "I was well until my periods began;" or, "My disease showed itself soon after marriage;" or, "I have never been in good health since my last labour, or since a miscarriage at such a time." That there were exceptions to this I need not mention, but they only appeared to be sufficiently frequent to prove the rule.

FOURTH CLIMACTERIC.—We come now to the consideration of the menopause, or change of life, which has been from time immemorial known as the technical "climacteric" of woman.

When the far-reaching nervous influences exerted by ovulation and menstruation upon the system of the woman, which continue active for thirty-five years of her existence, are borne in mind, one is prepared to expect decided mental and physical manifestations when a sudden cessation of ovular activity occurs about the fiftieth year. And certainly at that time the nervous system of the woman does become especially liable to loss of balance, as demonstrated by hysterical and nervous manifestations, and even by a tendency to melancholia and other psychic derangements; while disorders of the menstrual flow become marked, decided, and troublesome, and serious tissue-changes in the pelvic viscera appear to be especially frequent. While all this is admitted it cannot be denied that too great stress has been laid upon this period of woman's life as a critical epoch, and that many pathological conditions occurring about this time have been credited to it which have had no connection with it at all.

The swing of the mental pendulum in reference to the matter must be carefully supervised as to both its extremes; the gynecologist being careful, on the one hand, to avoid an over-estimate of the importance of this epoch which leads to making it a shield for incapacity or ignorance; and on the other, being wary of an under-estimate, which may cause him to lose the great advantages which may result from a postponement of operative interference until its occurrence be allowed to exert its legitimate beneficial influence. Indeed, I feel that I stand upon defensible ground when I assert that the chief significance of the menopause

to the gynecologist exists in the fact that it constitutes a haven of rest for the woman from many ailments which are due to menstrual disorders and to hypertrophy and hyperplasia of the uterus. It ordinarily puts an end to subinvolution and uterine hyperplasia, to menstrual irregularities, to metrorrhagia, and to uterine myomata. With such certainty can these results be counted upon that operations for such conditions may be held in reserve, in the hope that by this climacteric the necessity for them may be avoided.

Some years ago, when the effects of galvanism in the cure of uterine myomata was upon the crest of the gynecological wave, a former pupil of mine, a man of great industry, enthusiasm, and honesty, requested me to read, prior to publication, a report of twelve or fourteen cases of uterine myoma in which small tumours had been completely cured, and large ones greatly benefited and checked in growth by this, then new, therapeutic resource. It was not a report of his experience with the method, but one embodying his successful cases. Upon a careful analysis, I found that all the cases of recovery and amelioration had occurred in women between 43 and 53 years of age, and that each recovery was coincident with cessation of menstrual flow and other signs of the menopause. The pleasure of reading this honest and laborious account of faithful work was never enjoyed except by the writer and myself, for it did not appear in print!

At the time of the menopause, as atrophy of the cervix uteri and atresia of the cervical canal take place, retention of discharges from the endometrium sometimes occurs, constituting hydro-metra, hæmato-metra, or pyo-metra, and when decomposition of the arrested fluids supervenes, the rare condition of physo-metra shows itself.

Two forms of senile vaginitis are apt also to develop themselves about this time—adhesive vaginitis, which closes the vaginal canal entirely, and hæmorrhagic vaginitis, which I have on several occasions seen mistaken for carcinoma uteri, on account of its causing sanguineous flow in a woman who has for some time ceased menstruating. In this connection this diseased state is of importance, for the patient will always come to the physician in the greatest state of alarm. Fortunate is it that this alarm may be quieted by the promise of complete recovery.

Lastly, at this climacteric procidentia of the atrophic senile uterus is apt to occur from rapid

absorption of adipose tissue in the pelvis, which causes it to descend from want of support, and inflicts upon the woman a disability at the end of her career, and after she has borne triumphantly the burdens of life.

This completes the list of the most important of the pathological conditions which develop in connection with the menopause, the fourth climacteric of woman's life, and constitutes all that I have to say upon this subject this evening.

THERAPEUTICAL NOTES.

The Fever of Phthisis in Relation to Prognosis.—Dr. Strümpell emphasizes the importance of recognizing the particular type of fever present in cases of pulmonary tuberculosis. He says that if one is to arrive at a correct appreciation of any particular case, special attention must be paid to the question of prognosis, a question in which the physical condition of the lungs plays but a subordinate part. On this point there are other far more important factors to be considered, such as hereditary taint, the general and constitutional necessities of the patient, among the latter of which Professor Strümpell considers chronic alcoholism to be the most unfavourable; the pace at which the disease has hitherto progressed; and finally the fever. As to the cause of the fever, Strümpell is of opinion that, except in miliary cases, it is not to be ascribed to the tuberculosis, but to secondary inflammatory processes, set up by the invasion of pus and other cocci. In a number of cases there is, as everyone knows, no fever at all. The patients feel well, their lung trouble being stationary or making but minimal advances. Should, however, fever set in, its type is of the greatest importance for establishing a prognosis. Strümpell classifies the various forms of fever in phthisis as follows: 1. *Status subfebrilis*; here the morning temperature is normal, the evening, 100.4° to 101.3° F. In such cases the disease makes but slow progress, and amelioration may be expected by improving the general condition of the patient. 2. *Febris hectica intermittens*. With this type of fever (morning nearly normal, evening, 101.3° to 104° F.) the disease is steadily advancing, though in such cases a feeling of good health may be

maintained for a considerable time. 3. *Febris remittens* (morning, 100.4° to 101.3° F., evening, 103.1° F.). This type is far more unfavourable, as it points to the presence of lobular inflammation. 4. *Febris continua*. Except in miliary tuberculosis, this form of fever is found almost exclusively in cases of phthisis which start with acute symptoms, though it may be found interpolated for several days in cases in which the fever otherwise runs a remittent or irregular course. In either case the prognosis is unfavourable. The last remark is equally true of Strümpell's fifth fever type, that totally irregular form which is observed throughout the whole illness in many cases, though only in the last stage in others. That exceptions to the above rules will be found goes without saying, but it is none the less a significant fact, that in nearly all cases of phthisis in which pyrexia is present, the fever invariably assumes the form of one, and only one, of the types in question. Any change which may take place is brought about by the presence of complications, and it is frequently observed that the fever, set up by intercurrent diseases, lasts on after they have subsided, thereby showing that they have instilled renewed vigour into the phthisical process. Granted that the fever forms a most important indication in prognosis, it follows that it must also be the standard by which antiphthisica must be tested, for it is only by their effect on the fever that a just estimate of their value can be arrived at. On these grounds Strümpell rejected both tuberculin and creosote in the treatment of phthisis; the former he considers to be absolutely injurious, having frequently seen apyretic converted into febrile forms by injections of Koch's specific.

(*Muench. Med. Woch.—New York Med. Rec.*)

Hay Fever.—Dr. Bishop proposes a new theory and new treatment for hay fever. Adopting the suggestions and facts furnished by Dr. Alexander Haig, he asserts that the disease is of uric acid origin, and that the paroxysms are brought on by the irritation of this acid, which gets into the blood in excess, and causes the nerve-storms of sneezing, coughing, etc. The cure for an attack is to take ten to thirty drops of dilute Sulphuric Acid two or three times a day. The first dose should be taken early in the morning, and should be largely diluted with water. In the intervals, or, more particularly, about four days before an attack is expected,

Salicylate of Soda and Phosphate of Sodium should be taken. A diet of milk, fruits, vegetables, butter, etc., should also be adopted.

Dr. Bishop does not say that uric acid is the only cause of hay fever, or that his treatment should exclude attention to nasal and other irritations, but by following it one causative factor that is often prominent, if not essential, is excluded.

(*N. Y. Med. Rec.*)

Dyspepsia and Constipation.—Germain Sée considers that most of the conditions treated as dyspepsia, dilatation of the stomach, etc., are really sluggish conditions of the enteric mucous membrane due to constipation. He insists on the mechanical rôle of this constipation in causing hæmorrhoids, hernias, vesico-uterine tumours, hypertrophy of prostate, etc. He recommends that the constipation be overcome without purgatives, but that simple laxatives like Senna, Hydrastis Canadensis, Castor Oil or large doses of Olive Oil, be used. To relieve the pain attending these conditions of the mucous membrane of the intestine, he recommends:

R. Calcium Brom.

Calcium Chlorid. ... āā gm.50

Aquæ gm.500

Sig. Dessertspoonful in twice its quantity of water at meal-times.

For particularly acute crises of pain, he uses:

R. Menthol. gm.10

Spt. Vin. Rect. q.s

Aquæ Des. gm.180

Sig. Teaspoonful as indicated.

To combat fermentation and tympanism, he recommends Phosphate of Soda ʒij to ʒiij, Salicylic Acid, gm.iiij to vi, and Borax. To combat auto-intoxication, he regulates the diet.

(*New York Med. Rec.*)

Muscular Rheumatism.—We lately gave a formula for the local application of Salicylate of Sodium in rheumatism. Dr. T. H. Manley, of New York, has improved Dr. Lausanne's formula, so that it now runs:—

R. Chloroformi Pur. ... 3v

Tr. Opii ... 3iv

Acid. Salicyl. ... 3iv

Spt. Vin. Rect. ... 3iv

Olei Dulcis ... ad 3xij

M. Ft. lñ, Sig. To be applied *ad lib.*

(*Med. Rec.*)

THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 21, 1894.

A CLINICAL LECTURE ON MITRAL OBSTRUCTION.

Delivered at the Middlesex Hospital, Feb. 2, 1894, by
SIDNEY COUPLAND, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—I propose to-day to speak of a rather common affection of which we often have examples in the wards, and of which there came into Murray Ward recently three cases, which some of you may have seen last Tuesday. It will, I think, be most convenient if I first of all give you a brief sketch of each of these cases and afterwards offer some comments on them, along with some general considerations with reference to the affection which they represent.

The first case is in bed 2 of Murray Ward. It is that of a young woman, aged 27, unmarried, who is very anæmic, and has been subject to what she calls "rheumatism" ever since she was a child of 6. Last year she had a severe attack of rheumatic fever, and was laid up for six weeks. On the 27th of January she came into hospital suffering from a recurrence of her rheumatism. A week previously she had felt pain in her right wrist, and afterwards in her knees. When she came into the hospital, however, although she was slightly feverish, her temperature being about 101°, there was no objective evidence of any arthritis except slight swelling about the right wrist; the knees were perfectly natural. She does not seem to have suffered from any other affection in early life. She is well nourished. Her father died of bronchitis, and was rheumatic; her mother is paralyzed.

The only point about this case is the condition of the heart. When she came in there was, as I have said, slight pyrexia. There was no cough, no pulmonary signs nor any subjective cardiac symptoms; but on examining her heart we found a well-marked thrill on palpation, which terminated in the shock of the ventricle, this thrill being felt in the region of the apex beat which was about the normal situation—perhaps a little outside—in

the fifth space almost in the nipple line. The thrill corresponded to a bruit of the same character that occurred before the systole, and terminated in an abrupt first sound. At the base of the heart both sounds were then clear. Of her other symptoms none were important. She was slightly constipated. There was no enlargement of the liver, and the urine was normal. That was four days ago; and at the present time these signs still persist. The thrill is still felt, especially when she sits up in bed, and the bruit, which is of typical character, *i.e.*, rough and increasing in intensity till it comes to an abrupt termination in the first sound, is still persistent. Besides that there is now distinct reduplication of the second sound, this reduplication being quite limited in area to the region of the apex of the heart. It is not heard over the pulmonary area, *i.e.*, over the third costal cartilage, nor to the right of the sternum at all. This reduplicated second sound gives the rhythm of the heart a somewhat cantering character.

The next case is in No. 6 bed—a married woman, aged 30, who has had two children, the elder of whom is 11, the younger 2, both healthy. She tells us that when a child of 7 she had chorea, which lasted on and off for three years. She has had other childish ailments: measles, whooping-cough, etc. A year ago she had rheumatism, without any definite febrile attack, and also influenza. Over and above that, for the last two years she has been troubled with some abdominal symptoms. There has been pain in the abdomen, and the abdomen has been getting larger. This commenced about the time her last child was born, when, too, she seems to have had an attack, so she tells us, of pleurisy on both sides; but it is very difficult to be quite sure of this. She also says she had "a kind of fit"; but this also is very indefinite, and we could not make out that it was anything of the nature of an epileptic attack. She "brought up a teacupful of blood;" this may have been either hæmoptysis or hæmatemesis—which, I cannot say. Her pain and discomfort continued, and last October she went to a special hospital for women, where they told her she had a tumour in the abdomen. In addition to this, she had been latterly getting short of breath on exertion, and suffering occasionally from palpitation of the heart.

With this increasing discomfort she came into the hospital on the 24th of January. We found that she was slightly jaundiced. There was no marked yellowness of the skin, but this tint was distinct in the conjunctiva. This would bear out her statement that she had something abdominal. Her abdomen was found to be enlarged—due to great enlargement of the liver, which extended below the level of the umbilicus and could be readily palpated. The liver felt very hard, its edge sharp and its surface smooth. There was evidence of a little ascites. There is still dulness in the right flank; but when she lies on her back there is no perceptible dulness in the left flank. There is, however, a more valuable sign of the presence of fluid than dulness of the flanks, namely, a distinct sense of displacement on palpation over the surface of the liver, indicating that some fluid intervenes between the abdominal wall and the surface of that organ. She has thus slight jaundice and slight ascites, both due probably to derangement of the liver. In addition, there is also some cutaneous dropsy, not of the legs; the feet, ankles and legs show no pitting on pressure; but there is distinct pitting over the lower lumbar region behind. She is still short of breath on exertion.

On examination there is no sign of bronchitis; no dulness at the bases of the lungs. The shortness of breath is not due, therefore, to any pulmonary disorder. The heart is slightly enlarged. The apex beat is in the nipple line in the fifth interspace. The cardiac dulness commences about the fourth rib, and extends to the mid-sternum on the right. At the apex a distinct double murmur can be heard—a systolic murmur followed by a diastolic, both very much the same in character, blowing and soft, but the diastolic is much fainter than the systolic; indeed, the systolic is almost musical; it is higher in pitch, and can be heard all over the præcordia. You can hear it most intensely at the apex, but it can also be heard at the base, and it is conducted round the axilla towards the back.

But this is not all; for, on moving the stethoscope upwards towards about midway between the nipple and the sternum, we found two murmurs of a different rhythm. It is very striking to compare the two areas, the one over the region of the apex, and that in this region above. The diastolic murmur is entirely lost in this upper region; in its place there is a distinct presystolic, followed by the systolic. This change of rhythm in the mur-

murs, which may or may not be due to actual difference in their origin, is very striking, seeing that you are listening over two points so close together. You have only to move the stethoscope one intercostal space to find this remarkable change.

At the present time her condition remains much the same. This morning the presystolic murmur seemed very rasping and prolonged; and I noticed, too, for the first time, a faint thrill over the præcordia, which did not seem to me to be purely presystolic.

The third case is that of a woman, aged 35 (bed 13, Murray Ward), who was admitted for the third time on the 10th of January. She has been in this hospital on two previous occasions. The first was in the spring of 1891, at which time she was bearing a child, and was, I think, confined here; the second in January, 1893. On both occasions she was in for the same condition: bronchitis, pulmonary congestion, and well-marked cardiac disease. She says she has been subject to bronchitis since childhood. Every winter she has had cough, and during the last nine years it has been particularly bad. In addition she has had palpitation and præcordial pain on and off for a long time. Still she manages to get about and perform her household duties. She has never had rheumatism or chorea, nor, indeed, any affection which is known to cause endocarditis. Her father is rheumatic and has had rheumatic fever.

Seven weeks before admission this time she was attacked with bronchitis, and then with some sharp, shooting pains in the region of the heart. Her legs and feet began to swell, and her breath got shorter,—there was, in fact, the typical history of a cardiac patient. We found her somewhat cyanosed, livid lips, capillary injection of the cheeks, which were also of a bluish tint, some anasarca of the lower limbs (not extreme, there being slight pitting as high as the knee) and slight ascites. She had also orthopnoea, and a feeble, small, but regular pulse-beat (100). The last time she was in the hospital her pulse was very intermittent. All over her chest were to be heard coarse rhonchi and a few rales, particularly at the bases; but there was no dulness at the base of the lungs. She had slight cough with some mucoid expectoration. The apex of the heart was in the fifth space in the nipple line. A very typical presystolic bruit terminating in the first sound was audible. The second sound was

distinctly reduplicated. The liver could not be felt; but there was some tenderness in the hepatic region below the ribs. She complained of headache and fulness in the head, a symptom which really distressed her more than anything. Her face looked congested, and her headache was probably due to the venous congestion; it was certainly relieved by the application of a leech behind the ear.

At present she is still somewhat cyanosed and is breathing at the rate of 40 per minute. Her pulse varies from 90 to 84; it is small but very regular. The bronchitic sounds are less appreciable, and there is now no dropsy at all. A well-marked presystolic thrill can be felt, and there is a typical, rough, presystolic bruit, heard best about one inch above the apex beat. The first sound is very short, loud, and accentuated, and is followed by a distinctly reduplicated second sound. Traced towards the axilla the bruit soon disappears. You cannot hear it much outside the apex; but the short, loud, first sound, though getting fainter somewhat, as one traces it, can be heard right round to the back. Placing the stethoscope over the xiphoid one hears no bruit there, and the first sound is by no means so accentuated, but has much more of its normal dull character. Traced upwards between the nipple and the sternum the bruit becomes shorter, but the accentuation of the first sound remains very marked, as well as that of the second sound which is not reduplicated there, but is almost of the same character, pitch, and duration as the first sound. These two sounds are certainly not due to reduplication of the second, their rhythm being systolic and diastolic. At the aortic cartilage the second sound is much less loud, and the first sound soft.

These three cases, then, which differ so much in their history and in their subjective symptoms, agree in one thing, namely, the existence of signs of mitral obstruction. The essential signs of this defect are two, the presence of a thrill and the presence of a murmur, which, of course, are practically the same thing, the murmur being the audible thrill. In each case thrill and murmur coincide, and, in the main, precede the systolic shock and sound.

The mechanism of the production of this thrill or murmur has, as you are doubtless aware, been the subject of much controversy, into which I do not propose to enter at any length. More than

the whole hour at my disposal might be occupied in the discussion of this question as to the actual mechanism of this remarkable murmur or thrill, because it is quite *sui generis*. Suffice it to say that the most generally accepted notion is that of Dr. Gairdner, one of the earliest to describe it, who called it "auricular-systolic," implying that the bruit is produced as the blood flows into the ventricle from the auricle; that it is, therefore, a direct murmur heard best over the place of its origin, and comparable to the direct systolic bruit of aortic obstruction, which is also best heard over the area of the chest in most direct relation to the aorta, and, therefore, to the aortic valve. The alternative view, argued with great ability, is that the bruit really is produced after the blood has entered the ventricle, that it occurs in the first period of the ventricular systole, and is, therefore, an indirect or regurgitant bruit homologous with the common systolic regurgitant murmur of mitral incompetence. I would only remark that on many grounds the former doctrine seems better supported by facts. First of all, we have the character of the murmur. It is of a coarse rough nature resembling that produced over the aortic valve in aortic obstruction by the passage of the blood through the narrowed orifice. That, of course, does not go for much, because it may be said that a murmur of this character would be produced whether the flow were in one direction or the other. Next, we have its localized area. That is practically limited to a spot nearly over the place of its production. It must, therefore, be due to the blood flowing through the narrowed orifice; but this, again, does not prove in which direction the current is flowing. More important in deciding the question is that it distinctly precedes the first sound of the heart and the ventricular-systolic impulse; therefore, it occurs in the diastolic period, a period corresponding to the auricular systole. And lastly, there is the fact which one can often prove, namely, its coincidence with the pulsation of the auricle. In these cases, where the auricle is generally enlarged, we can see it pulsating in the third space. We see an impulse there which alternates with the ventricular impulse, and that pulsation above in the third space is contemporaneous with the murmur and thrill. That thrill is, perhaps, the strongest proof, if it were needed, that the murmur is truly auricular-systolic.

The method in which the bruit is produced

whether by the blood flowing in one direction or another, is, after all, rather an academic question; the essential thing is that it is an index of mitral obstruction in the vast majority of cases. I say "in the vast majority of cases," because there is no rule without an exception; and so it is here. Presystolic murmurs have been heard, and presystolic thrills have been felt where subsequent examination has shown the mitral valve to be quite normal. The number of such cases, I believe, could almost be counted on the fingers of one's hand. I cannot say I have ever noticed it myself; but the late Dr. Austin Flint has recorded one, Dr. Sansom another, and Dr. Lees another, where, as I say, the mitral valve was found to be healthy, although, during life, there were these signs which have been thought to be pathognomonic of mitral stenosis; and in those exceptional cases aortic regurgitation was the lesion found. Yet this has its parallel in the converse exception. It is exceptional to hear in mitral stenosis a diastolic murmur without there being any aortic regurgitation. So that we have this curious blending of facts—on the one hand we have the common murmur of mitral stenosis, a presystolic murmur, being rarely heard in cases of uncombined aortic regurgitation, and on the other we have the common murmur of aortic regurgitation,—the diastolic—being heard sometimes where mitral stenosis is the sole lesion.

I may briefly remind you of the difference between these murmurs, illustrating my remarks by these diagrams. Here, for example, is represented a typical auricular systolic (*i.e.*, presystolic) murmur. The murmur may be only in the latter half of the diastole, and running up to and abruptly terminating in the first sound; or it may be much prolonged, starting close upon the second sound, and running up through the whole diastolic interval. On the other hand, all pure diastolic murmurs terminate before the first sound. The exception I have mentioned is here represented—a diastolic murmur not actually coming off from the second sound, but still occupying the diastolic interval and terminating before the first sound. The existence of such anomalous cases seems to show us how important it is to pay attention not merely to the cardiac auscultation in making a diagnosis of a valvular defect, but also to the concomitant elements in the case. You know what a valuable sign of aortic regurgitation is to be found in the character of the pulse; so that whether you

have an ordinary diastolic murmur or not, if the pulse shows evidence of aortic regurgitation, you would trust that rather than the auscultatory signs. So too with the other auscultatory symptoms to be taken into consideration in diagnosing between mitral and aortic disease. There are two characteristic auscultatory signs in mitral stenosis, namely, the accentuation of the first sound, and the accentuation and reduplication of the second. As to the first of these—the accentuation of the first sound—it is quite as striking a feature of mitral obstruction as is the presystolic murmur. I might even say that it is the more constant feature, because there are cases of undoubted mitral stenosis where the murmur is absent, and only the short, sharp first sound is left. As the mitral stenosis increases the conditions for the production of the murmur seem to become less marked. Perhaps the chink is reduced to so small a size, that there is hardly any vibration as the blood passes through it; but still the short, sharp first sound remains, and you may sometimes have to diagnose mitral stenosis from that one fact. In an ordinary case, such as one of these I have read, this sound is very loud—in fact, it is almost painful to listen to. Its abrupt termination, as well as its sharpness and loudness, contrast markedly with the softer tone of the normal first sound of the heart.

I may here remark that we have another instance of a short first sound in the case where the muscular wall is weak, apart from any valve defect, but that has not the same intensity as the sound in mitral stenosis. No wonder, then, that in the early days of cardiac auscultation, this short sound was regarded as the second sound of the heart. By moving the stethoscope, however, and listening to the rhythm of the two sounds, there is no difficulty in distinguishing it. It is heard with greatest intensity where the bruit is best heard—usually within the nipple, also at the apex, and round the axilla to the back, losing in its sharpness and loudness somewhat. In the case in Murray, 13, this is particularly noticeable. It is suggested by Dr. Sansom that this sound is really a tricuspid and not a mitral sound; and there is great force in this view, for in advanced mitral stenosis, where the valves are fused into one, for instance, it is hardly conceivable that there should be any first valvular sound produced there, whereas, in such a case there is sure to be greater strain upon the right ventricle, and the tricuspid valve will probably

close with greater force. I think it is probable that this is the true explanation; but my only difficulty in accepting it is exemplified in such cases as that now in Murray, 13, where the loud sound is best heard at the apex of the heart, over the area where the presystolic murmur is heard, and out towards the axilla; but when you listen over the xiphoid and over the tricuspid area itself, the sound is found to have lost in its accentuated quality. This may be only one case out of many which would run counter to this ingenious hypothesis, or again, it may be quite exceptional.

As to the second sound, there is no doubt that the pulmonary valves are more sharply brought together by the increased force of the right ventricle, and the backward pressure on the pulmonary circuit. It is also generally believed that the reduplication is due to want of synchronism in closure of the aortic and pulmonary valves.

That is the accepted notion—that when one gets increased tension upon the pulmonary circuit, the valves close sharper and quicker than those in the aortic, and *vice versa*. But here again we owe to Dr. Sansom's admirable critical faculty another explanation which possibly may be a truer one. These two second sounds, he thinks, are not due to want of synchronism in the closure of the aortic and pulmonary valves; indeed, he says one of these elements is not a second sound at all, but is concerned with the thickened mitral valve. In support of that, I may say, is the fact that, as a rule, we do not hear this reduplication in mitral stenosis over the area of the valves themselves, *i.e.*, over the third costal cartilage, where you are listening exactly over the site of the aortic and pulmonary valves; but we do hear it best where we hear the murmur best—lower down, nearer the apex beat. Two of our cases illustrate this well. What is heard at the pulmonary area is the single loud pulmonary second sound. There is no question that this loud pulmonary second sound is a valuable sign in mitral stenosis; but reduplication is only to be heard over a limited area in and about the region of the apex beat. Now and then one may get a localized diastolic bruit over the pulmonary area, in association with obstruction at the mitral orifice. This is a very interesting thing, because it suggests the existence of the rare condition of pulmonary regurgitation, and there is every likelihood that such does occur. Where you have increased pressure upon the pulmonary circuit, it may sometimes happen that

the artery yields, and dilates so that the valve is rendered incompetent; and a short, soft diastolic murmur is to be heard there in some of these cases of mitral stenosis.

Leaving these interesting, and, to a certain extent, speculative questions, let us now inquire into the pathological conditions which underlie these signs of mitral obstruction. These may be put under two heads.

1. Vegetations on the mitral valve in recent endocarditis. These are quite sufficient to produce the evidences of mitral obstruction that I have mentioned. Even in cases of acute rheumatism and chorea they may develop, or wherever there is acute endocarditis. We had a good example of that the other day in a patient with kidney disease, accompanied by what are called signs of mitral stenosis, but which we prefer to call signs of mitral obstruction. There was a presystolic thrill and bruit. After death we found a mass of vegetations on the valve. There was no stenotic contraction of the mitral orifice, strictly speaking, merely a mass of vegetations.

2. The more common condition associated with these signs is that of fibrosis or sclerosis, where the valve cusps get fused together and greatly thickened. This thickening spreads down to the chordæ tendineæ, many of which get fused to form thick bands, and the muscoli papillares get capped with tough fibrous tissue. There are two kinds of contraction produced in this way; and it is these two classes which are strictly and properly called mitral stenosis. The first is that in which the mitral valve is converted into a sort of diaphragm with a slit in it—a button-hole slit. This slit may be very narrow indeed, the two thick cusps standing out horizontally with the narrow button hole between. In the other form the cusps are fused together to form a sort of funnel with a hole in its apex. The "button-hole" mitral and the "funnel-shaped" mitral, then, are the two kinds produced. No doubt there is some difference in the mechanism of their production, but I cannot attempt to explain that. It is very natural to think that the funnel shape is produced by a simple hydrostatic action of the blood distending the auricle and pushing the damaged and inelastic valve down.

But how is it that the valve becomes changed and distorted in this remarkable way? *A priori*, of course, it seems difficult to believe that vegetations in pure endocarditis should lead ultimately

to this remarkable change in conformation and structure; and in a number of cases there is no primary history of acute endocarditis, and no history of any disease which we know to be associated with acute endocarditis; but in others just as typical there is, and it seems more likely

have acute endocarditis associated with these chronic changes; but that is an epi-phenomenon, what is called ulcerative endocarditis, which, indeed, favours chronically affected valves of this sort. But, I believe, these cases of mitral stenosis are chronic, as it were, from the beginning—a

TABLE A.
ANALYSIS (CLINICAL) OF 150 CASES OF VALVULAR DISEASE.

				MITRAL ALONE.			AORTIC ALONE.			MITRAL & AORTIC COMBINED.		
				Regurgi- tation.	Obstruc- tion.	Reg. & Obstr.	Regurgi- tation.	Obstruc- tion.	Reg. & Obstr.	Regurgi- tation.	Reg. & Obstr.	
Males	21	2	5	1	...	7	21	15	72
Females	23	6	25	2	...	3	7	12	78
Total				44	8	30	3	...	10	28	27	150
Previous History of Rheumatism.				27 or 61.4%	3 or 37.5%	20 or 66.6%	1 or 33.3%	...	2 or 20%	14 or 50%	14 or 51.8%	81 or 54%
Fatal Cases	{	Males	...	5	1	...	1	...	1	8	4	20
		Females	...	5	3	3	1	1	2	15
Total				10	4	3	1	...	2	9	6	35
Mortality Rate				22.7%	50%	10%	33.3%	...	20%	32.1%	22.2%	23.3%

TABLE B.
ANALYSIS OF NATURE OF LESION OF MITRAL AND AORTIC VALVES IN 24 CASES OF VALVULAR DISEASE, WITH THE KIND OF MURMURS NOTED DURING LIFE.

LESION.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Post-mortem Examination.
Sclerosis	M	...	M*	M	...	M	...	M	M*	M	M	M	...	M	M	M*	M	A*	
Sclerosis + Veg. ...	A*	A	M*	M*	...	M	A	Clinical Signs.
Sclerosis + Calcif...	...	A	A	...	A	A	A	M	A	
Vegetations	A	A	M	...	M	...	A	A	M	Clinical Signs.
Ulceration	M	...	M	M	
MURMUR.	† †																								Clinical Signs.
Apex Presystolic	1	1	1	1	...	1	...	1	1	...	1	
Apex Systolic	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	Clinical Signs.
Basic Systolic	1	1	1	1	...	1	1	1	1	1	1	1	1	...	1	...	1	1	
Basic Diastolic	1	1	1	1	1	1	1	1	1	...	1	...	1	...	Clinical Signs.
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	

* Indicates that the valve-orifice was stenosed.

† Previous history of rheumatism.

that the change is mostly a progressive one in the substance of the valve, akin rather to the slow progressive changes in the substance of the liver in cirrhosis, or in the kidney in its contracted granular form, than to a secondary affection of acute endocarditis. We do, indeed, sometimes

slow, progressive, fibrotic change going on in the valve.

In connection with this I wish to refer to these diagrams constructed some years ago from an analysis of a number of cases of disease of the heart. They comprise the statistics of 150 cases

of valvular disease of the heart recorded in my case books (1876-1886). The cases are grouped under three heads, (a) mitral disease alone; (b) aortic alone, and (c) aortic and mitral regurgitant combined. This (Table A.) is a clinical table; below we have the pathological table (Table B.), comprising those cases that came to the post mortem room, and in which we were, therefore, able to determine the precise nature of the lesions of the valves.

Assuming the diagnosis (based on auscultation) to have been right in the first group, we found that, as regards mitral disease there were twenty-eight males to fifty-four females; whereas, in combined mitral and aortic disease, there were thirty-six males to nineteen females; and in simple aortic disease eight males to five females. Thus the relative frequency of mitral and aortic disease is reversed in the two sexes.

Another thing to be noticed in that table is the rarity of uncombined obstructive disease. There are none of aortic, and only eight of mitral stenosis in this category. This shows how seldom obstruction occurs apart from regurgitation. This is only natural, because if the valve be so damaged as to cause marked obstruction it is sure to be also incompetent. The rarity of aortic disease, apart from mitral, is also shown. I need not go into that, but I may remind you that mitral disease often follows upon aortic. The mitral may not be originally (primarily) affected at the same time as the aortic valves, but may become secondarily incompetent from dilatation of the ventricle. I have separated the cases of aortic disease complicated with simple mitral regurgitation from those in which the combined mitral lesion was obstructive or double. The two classes were nearly equal. In the former there is no actual disease of the mitral valve, no mitral endocarditis. In the other cases where there was evidence of mitral stenosis, there is certainly such mitral lesion. When you get mitral regurgitation associated with aortic disease alone you may have a great many without endocarditis, but you never get mitral stenosis apart from structural lesion.

With regard to the question of rheumatism, the table gives the total number and the percentage of frequency in each class. This might have been further analyzed to include only those cases in which there is a history of one or more attacks of rheumatic fever; but here are included also even those cases which had a history of very slight symp-

toms of rheumatism, numbering 54 p.c. of the whole series; with mitral disease 60 p.c.; with simple aortic 23 p.c.; with combined mitral and aortic 51 p.c.;—the broad inference being that people with mitral disease have more frequently a rheumatic history than those with simple aortic.

In contrasting the relative rheumatic history and mortality of different forms of valve lesion, it may be observed that mitral obstruction stands low in the scale as regards a previous rheumatic history, but very high as regards fatality.

One point of special interest with regard to mitral stenosis is the preponderant liability of the female sex. This is balanced by the preponderance of purely aortic stenosis in the male; but there is no parallel, of course, because in aortic disease males are subjected to greater arterial strain. This difference in the incidence of mitral and aortic lesions with regard to the sexes has attracted a good deal of attention, and several explanations have been offered. One, Dr. Goodhart was responsible for, namely, that anæmia and chlorosis in early life might be the starting point of the process in the mitral valve. Dr. Newton Pitt, who analysed the post-mortem examinations at Guy's Hospital, found an unusually large proportion of examples of granular kidney in people with mitral stenosis; but when we remember that women are not so liable to gout and granular kidney as men, that does not, I think, help us much, seeing how comparatively rare is pure mitral stenosis among men.

As to the effect of the lesion, and therefore the *prognosis*,—in these three cases there are probably represented three different degrees of the lesion. In the woman in bed 2, the first case read, the disturbance is comparatively slight; she has no cardiac symptoms; there is ample compensation. She came in, not because she had mitral stenosis or mitral obstruction, but simply because she had some rheumatic pains. The patient in bed 13, who has had bronchitis for so many years, is obviously suffering from a far more advanced lesion. Her long history of bronchial catarrhs indicates the long duration of her valvular defect. This is her third appearance in hospital, and the dropsy and cyanosis prove that compensation is failing.

The case in No. 6 is less clearly uncomplicated,—the patient with the big liver. I am not yet quite convinced that other valves are not involved in the same change as the mitral. Here are specimens showing simultaneous thickening and narrowing of

the aortic, mitral, and tricuspid valves; and the lesion may have attacked all the valves in this case. There is, in addition to the presystolic, a diastolic murmur at the apex, besides which there is the enormous enlargement of the liver, which is uniform, and is, I think, due to chronic venous congestion. It certainly is a clinical and pathological fact that in some cases of mitral disease, particularly stenosis, all the stress of the disturbed circulation may be felt upon the abdominal organs rather than on the lungs. You may have ascites without other dropsy, but the ascites is really produced through the liver, and is due to portal obstruction brought about, not in the ordinary way by cirrhosis, but by hepatic congestion. In this case we have, I think, a patient whose lungs have escaped, unless, indeed, the blood brought up two years ago indicated hæmoptysis. In the slight albuminuria there is evidence of renal congestion, which somewhat further modifies the prognosis.

In comparing the three cases one would say the prognosis is worst in the woman who has had bronchitis so long, and whose heart is so obviously failing now. One would say she has not many years to live. The next is that with the enlarged liver and albuminuria, and the most favourable prognosis would be in the case of the youngest woman.

The duration of life in people with this heart lesion is comparatively long. There is no doubt that in all these cases the lesion began in early life; so that you must not think when you find a girl with a loud presystolic murmur, or signs of mitral stenosis, that she will not grow up to womanhood. She may do so, but she may not reach the climacteric. Before then heart failure will set in, particularly if she have married and borne the strain of child-bearing, compensation will no longer operate, and she will have dropsy and die. That is the usual history of these cases. They come into the hospital when the condition is advanced, between the ages of 30 and 40.

But these people with mitral stenosis are also liable to other accidents which may cut them off before that time. Early in the session in the same ward, we had a woman suffering from mitral stenosis, who was brought here because of an attack of hemiplegia, due to cerebral embolism—probably from an embolus carried from the blood coagulum which had formed in the auricular appendix. The liability for such clots to be swept into the brain and elsewhere is great, and may cut

short the patient's life. Similar impaceptions may occur from clots coming from the right side of the heart, where also stagnation of blood is likely to occur. Infarctions of the lungs may thus be produced. Fresh attacks of endocarditis are also liable to occur of the ulcerative and malignant form, more like pyæmia than anything else. There are many dangers in the way of persons with chronic heart disease over and above those simply due to the valvular defect.

I have but little time left to speak of the *treatment* of these cases. The mere presence of mitral stenosis or of the signs of mitral obstruction does not mean that the patients have to be medically treated. No drug can be expected to restore the damaged valve, and under the conditions in which the valves act, the affection is bound to go on increasing; but careful management of the diet and a life passed in a healthy and rational manner, may carry these cardiac subjects through for a long time, and they may have no symptoms of the deranged valve mechanism, until the heart-muscle begins to fail. When they have to be treated, it must be as rationally as possible. The venous congestion from which they suffer must be relieved, by frequent slight purgation, with salines for instance. Hydragogue purgatives are sometimes useful; and sometimes venesection may be employed. If the heart be obviously failing, of course cardiac tonics are indicated. Digitalis, Strophanthus, Strychnine, Arsenic, are all of value to restore the healthy tone to the muscle and re-establish compensation. All the symptoms dependent upon the venous congestion will, of course, be combated if we can relieve that which underlies it, namely, the distension of the right heart.

What should be the guide in regard to stimulants? The longer I live the more I think that alcohol ought to be given very sparingly indeed to people with chronic cardiac disease, and one great consideration is that having once begun to give it in such cases it is very difficult, if not impossible, to leave it off. After all, the mere stimulant effect of alcohol is quite temporary; and it is highly probable that the continued administration of it is obnoxious in that it tends to diminish the desire for food, and perhaps may actually aggravate the tendency to induration, arterial and valvular, that already exists. Alcohol should certainly be sparingly given. So too with food. Not one of the least services rendered by the late Sir Andrew

Clark was his enforcement of the doctrine of spareness of diet in people suffering from heart disease. He pointed out the fallacy of the notion that people who "feel" weak have necessarily to be "supported" with highly nourishing articles of food and wine. Such is the reverse of rational treatment and sure to make the patient worse, adding to the difficulties of the labouring heart and so increasing all the symptoms. One, over and over again, sees the great relief afforded to these cases by reducing to the least possible amount the food given.

A CLINICAL LECTURE ON UMBILICAL HERNIA.

Delivered at King's College Hospital, Feb. 12, 1894,

By **WILLIAM ROSE, F.R.C.S.,**

Professor of Clinical Surgery, and Senior Surgeon to the
Hospital.

GENTLEMEN,—An aggravated case of umbilical hernia in a middle-aged woman, which I recently treated by operation, will form the subject of our clinical lecture to-day, and the basis of a few remarks on umbilical hernia generally. The following is the history of the case :

S. W., æt. 48, married, was admitted into the Wigram Ward on Jan. 4th, 1894. The following facts in the family history are interesting and worthy of note. A brother suffered from congenital inguinal hernia on the right side, and the patient recently had a son, aged 14, in the Albert Ward with an acquired right inguinal hernia, on whom I operated, and the lad has now left the hospital. She has also two other children, but they are not ruptured. Seventeen years ago after a severe labour she noticed a protrusion on the left side of the umbilicus, and attributed it to defective treatment after delivery. This swelling increased steadily in size, in spite of the use of a belt and pad, until it attained the dimensions shown on admission. At first the tumour was reducible, but soon it became fixed and irreducible, and recently she has suffered constantly from colicky pains, nausea, and irregularity in the action of the bowels.

On admission the patient, who was an exceedingly stout, florid and flabby woman, weighing nearly sixteen stone, was found to have in the um-

bilical region a large hernial protrusion eight inches in diameter, the umbilical cicatrix being situated about one inch from the upper margin. The skin over the swelling was thin and dimpled from evident adhesion to the structures beneath. A tympanitic note could be elicited in various parts, whilst others felt doughy and nodulated. There was great tenderness, particularly about the lower part; the bulk of the swelling could not be appreciably diminished by taxis. Urine 1030, with no albumen or sugar.

The patient was placed for a short time on an anti-fat diet, in which farinaceous food and carbohydrates were reduced to a minimum.

The operation performed on Jan. 12, 1894, consisted in laying open the loose bag of integument which might truly be said to form practically the sole covering of the protruded viscera, and in carefully dealing with the innumerable adhesions which existed not only between the wall of the cavity and the contents, but also between the coils of intestine and omentum which were of so dense a nature as to repeatedly necessitate division between double ligatures. Only those of you who were present can appreciate the unusual difficulties that we met with; the whole of the transverse colon, the omentum and coils of the small intestine several feet long were all matted together in a state of confusion that baffles description, and it was only by the exercise of the greatest care that we avoided tearing some part of the gut. After these adhesions had been at last successfully dealt with, and 1 lb. 12 oz. of omentum, granular and altered, had been removed, there still remained the difficulty of returning so large a quantity of bowel, particularly of the large intestine into the abdominal cavity. This was only effected by slightly increasing the opening in the linea alba in an upward direction with a probe-pointed bistoury. The exposed parts were throughout the operation protected by towels wrung out of hot carbolic lotion (1 in 60). The hernial aperture was next brought together in the vertical direction by a series of strong silk and catgut stitches, the loose redundant abdominal skin was clipped away with scissors, and the integumental wound closed by a continuous suture. The operation lasted one hour and fifty-five minutes. The after-progress of the case was eminently satisfactory, although a slight pocket of pus collected underneath the integument at the upper angle of the wound, probably from retained blood.

Such, Gentlemen, is an unusually severe example of a neglected umbilical hernia in an adult. Let us now pass to a consideration of the different forms of umbilical hernia, their method of production, and the means that must be taken to prevent them from attaining such formidable proportions as met with in the case under notice, and the reasons which induce us to advise operative interference.

The umbilical cicatrix, we must remember, represents the point of junction between the embryonic structures of the cord and those of the abdominal parietes. Their incorporation, however, is so intimate that the peritoneum, transversalis fascia, and aponeuroses are all blended together into one cicatricial mass which, under normal circumstances, is stronger than the surrounding parts. When, therefore, a hernia occurs in the adult, the protrusion takes place at the side of this cicatrix, and usually at its upper margin (probably owing to the closer approximation of the recti muscles below). On the other hand, it is to some faulty condition of these embryonic structures, that the so-called *congenital umbilical hernia* is due; for instance, when the intestines are not entirely withdrawn into the abdominal cavity, a stage of development which is usually completed at the fourth month, then a protrusion occurs for a short distance into the substance of the umbilical cord, and if unfortunately this escapes the notice of the practitioner or midwife, and the ligature is applied too close to the abdomen, the intestine will be strangulated when the cord is tied. A fatal result from peritonitis may follow, or at the best a *fecal fistula* will be established. Again, even when such a condition is recognized at birth, unless prompt operative measures are undertaken before the cord separates, septic infection of the peritoneal cavity is certain to follow that event. Early laparotomy is indicated, the protruding portion of gut reduced, and the aperture in the abdominal wall closed by suture. Fortunately, this malformation, though exceedingly rare, is easily recognized by the inflated condition of the base of the umbilical cord.

Another form of hernia is that which occurs shortly after birth, in consequence of a stretching and yielding of the umbilical cicatrix, a condition commonly known as "starting of the navel," or *infantile umbilical hernia*. This is an affection frequently met with, and is the result of increased intra-abdominal pressure, whether due to the child straining, or crying, or ineffectual efforts to empty the

bladder or bowels. You should bear in mind that the presence of a long foreskin with a pinhole preputial orifice is often associated with these cases. The usual treatment by pad and bandage is in the large proportion of cases sufficient to produce a cure; a small coin is wrapped up in a piece of lint and applied so as to exactly cover the protusion, and is fixed in position by strapping, the whole appliance being covered by a flannel bandage or binder. There are instances, however, which continue in spite of such treatment, the hernia persisting and even tending to increase; for such, a radical operation is indicated. The integument and sac are laid open by a vertical incision, great care being taken not to wound any of the subjacent viscera; this is more likely to happen if the patient is vomiting or coughing at the time the incision is made. The margins of the umbilical opening, having been clearly defined, are brought together in the vertical direction by silk or catgut sutures, and the integument treated in the usual way. Provided perfect asepsis is maintained, there is no risk, and the results are quite satisfactory.

Thirdly, we have the *acquired* form of hernia which is met with later in life, and to this, women who have borne children are particularly prone. In these the umbilical cicatrix does not itself yield in the first place, but rather a weak spot forms in the abdominal wall in the immediate vicinity, and through this parietal peritoneum and a portion of omentum or a coil of intestine are protruded. After the tumour has reached a certain size, the coverings become increasingly attenuated until there is merely peritoneum and integument remaining. A somewhat parallel condition is seen in a large sacculated aneurism, in which the coats of the vessel cannot be traced far from the mouth of the sac. Even the parietal peritoneum itself may rupture, or be attenuated to such a vanishing point that the protruded intestines and omentum are, so to speak, extravasated beneath the skin. This may account in some cases for the intimate adhesions which are found between the integument and the viscera. The integumental dimple of the umbilical cicatrix is generally stretched and lifted away from the aponeurotic portion, although a slight fibrous connecting cord may remain.

The *contents* of an umbilical hernia are usually intestine and omentum, but surgical curiosities are recorded where the gravid uterus and the stomach have been found in the sac.

When a protrusion of this kind occurs in this region, the patient herself endeavours to return the mass into the abdomen, or perhaps by the instructions of the doctor, in order to give herself ease, and an abdominal belt with an air- or water-pad is usually worn. In a short time, however, it is found that a certain amount of *inflammation* of a low type has been lighted up, partly by these efforts at reduction, partly by the inefficient action of the pad and the friction of the clothes. As a result of this, adhesions occur which render the tumour more or less irreducible. These adhesions form not only between the contents and the sac wall, but also between neighbouring coils of intestine and omentum, matting them together and thereby producing an insuperable bar to reduction by preventing those contents being returned *seriatim* through the small abdominal aperture. These recurrent attacks of inflammation are marked clinically by excessive tenderness, dragging pain, and often some nausea and vomiting. Peristalsis of the contained intestine is checked, and it would take but little to induce a condition of obstruction which might even lead on to strangulation. Where omentum only is present in the sac, it is possible that such an attack may lead to an imperfect natural cure by the plugging of the abdominal aperture with the omentum so as to prevent any further escape of the viscera. I say imperfect, because the fact of an adherent plug of omentum being present is a source of constant pain and discomfort to the patient, by interfering with the regular action of the bowels, as well as rendering the patient liable to internal obstruction in a variety of ways, the mechanism of which I must describe at another time. Such plugging of the abdominal aperture does not often occur, and though some adhesions may be present around some portion of the margin of the abdominal aperture, there is usually sufficient patency to allow of the passage of fresh coils of intestine—the result of unwonted and sudden exertion of the patient—which by the increased pressure induced within the sac leads to *acute strangulation*. This is not the only complication that a patient with a large umbilical hernia is subject to. Masses of partly digested food are liable to become retained in the protruded intestine, and set up symptoms of obstruction, colic, nausea and vomiting, which closely simulate those of a subacute strangulation, and may be the precursors of gangrene of the gut. At the same time, it is wonderful what rest and

judicious treatment will do to arrest so serious a termination. The patient is kept in bed, his diet restricted to slops, frequent and large enemata are used in the first place, followed by a dose of Castor Oil, when the more acute symptoms have subsided.

Into the subject of strangulation we cannot now enter, but one more complication must be alluded to in conclusion. We have recently had a case in the male ward of a stout man with a large irreducible omental umbilical hernia, in which the skin over the hernia became ulcerated and then infected by some virulent organism, setting up an acute cellulitis which threatened at one time to lay the sac open. The sloughing of the skin was most extensive, but a barrier of granulation tissue at length formed of sufficient strength to shut off the spread of the mischief to the peritoneal cavity, and the patient finally recovered with the hernia reduced to a minimum by reason of the cicatricial contraction which followed. Such a severe condition is not likely often to be met with, but simple ulceration of the skin is common enough, owing to defective nutrition, and needs treatment on general principles.

I think, Gentlemen, I have said enough to convince you of the dangers to which a patient is exposed who possesses a large irreducible umbilical hernia, and that inasmuch as no efficient support can be obtained to prevent the intrusion of fresh contents, there is every reason to adopt operative measures for the radical cure of such cases.

Sub-Gallate of Bismuth.—This remedy (Dermatol) has been found to be, in ten-grain doses after meals, almost a specific in cases of purely functional dyspepsia and flatulence.—FLINT.

(*New York Med. Rec.*)

Disinfection of Stools.—Dr. H. Gross strongly recommends Saprol for the purpose of disinfecting stools and urine. His especial reason for this is as follows:—All other disinfectants, when mixed with watery substances, sink to the bottom on account of their specific gravity, but Saprol, on the contrary, floats on the water, and thus disinfects primarily that portion of the mixture which comes in contact with the air. For disinfecting mixtures of urine and *fæces* only 1 p.c. Saprol is required, and its cost per head per year is a little under fourpence.—(*Fortschr. der öff. Gesundh.*)

A LECTURE
ON
OZÆNA, OR ATROPHIC RHINITIS.

Delivered at King's College Hospital, Feb. 21, 1894.

By GREVILLE MACDONALD, M.D.,

Laryngologist to the Hospital, and Physician to the Hospital
for Diseases of the Throat, Golden Square.

GENTLEMEN,—One of the most remarkable affections in all pathology is that on which I am going to speak to you to-day, namely, what is commonly termed ozæna, or, somewhat more precisely, atrophic rhinitis—although the latter is a term which does not completely describe the condition. I say it is one of the most remarkable diseases with which I am acquainted, on account of the total absorption of large masses of bone, and that without any of the usual forms of disease which we should expect in such a condition of affairs. There is no necrosis, no caries, and no formation of masses of granulation tissue; but slowly, imperceptibly, unreasonably, one might almost say, the whole substance of the inferior turbinated bodies, mucous membrane, erectile tissue and bone disappears, leaving not the slightest vestige of its normal condition. It is because of this absorption, this atrophy of the bone, that the condition is commonly called atrophic rhinitis; yet I shall presently adduce reasons for questioning the rectitude of the term.

There is very little I can tell you positively about the pathology of this disease, because very little is known. Different investigators have made different observations, and one contradicts another to such an extent, that it is scarcely worth while quoting any of them. The very process which we wish to investigate produces destruction so complete, and the patients so seldom come under observation until the atrophy is almost entire, that it is extremely difficult to determine in what that process consists. By the best observers, however, the mucous membrane is described as being replaced by large masses of round and fusiform cells of very indefinite outline, the substance of the cells being replaced by a fatty detritus. Here and there epithelial cells are found undergoing the same fatty degeneration, and there are also found larger or smaller accumulations of fatty globules. But even if such a description applied to every case, it would not explain matters at all; it would not

account for the process of absorption. Yet presently, when speaking of the etiology of the condition, I shall make a suggestion which may partly, or wholly explain the atrophy of mucous membrane and bone.

These cases of atrophic rhinitis, or, as I prefer to call it, ozæna, because the stench in the nose is the one characteristic symptom, occur for the most part in young females. Out of 100 cases, I found that 65 were women, and only 35 men. It is an affection of the period of early adult life. The average age at which my patients presented themselves for treatment was 18; but they seldom consult the specialist until the disease has been existent for a long time, although often enough they have had treatment from the family doctor. The disease, therefore, we may assume, begins considerably earlier than that—I believe about early puberty. It is more prevalent, I think, amongst the lower classes, who are badly fed and imperfectly cared for, although a considerable number of cases occur amongst the well-to-do. We find also that the so-called strumous diathesis (whatever that may mean) predisposes to the production of the disease.

The physiognomy of these patients is highly characteristic. It is the physiognomy that we commonly associate with the strumous diathesis. There is the prominent brow, the depressed point of origin of the nose, which is of a pug shape, the *alæ* looking downwards and forwards. The member is altogether curiously depressed into the cheek, as if the whole structure had shrunk in. Then the lips are a little prominent and deficient in expression: the general aspect being wanting in the accepted indications of general intelligence. But the point I wish to draw special attention to is the shape of the nose and the angle it forms with the brow. Looking at the full face, in the vast majority of these cases we find the bridge of the nose peculiarly wide; and as a matter of fact these patients begin life with abnormally wide nasal fossæ—the very condition which the disease tends to aggravate. While talking of physiognomy I may mention a point which anyone who has seen a large number of cases of nose disease will admit to be fact, that this type of nose—the small turned-up nose with a wide bridge and *alæ*—has a tendency to atrophic disease, a tendency to disease which will make the nasal fossæ still wider than they naturally are. But, on the other hand, the prominent nose, whatever its style, is generally the

nose with narrow nasal fossæ; and the latter condition predisposes indubitably to hypertrophic affections in one form or another. It is in such a condition that we are apt to get hypertrophy of the inferior turbinated bones, polypus, etc. And you will at once notice that the race, which as we see it in this country, has the most prominent nose, viz., the Hebrew, presents among the majority of its members more or less tendency, in popular parlance, to "speak through the nose;" their speech has the intonation of nasal obstruction, they speak as if with a chronic cold in the head. These are the noses which for the most part present opposite characteristics from those of the type now under our consideration.

I believe that this abnormal patency of the anterior nares actually does predispose to atrophic disease, for occasionally, though rarely, we find the affection unilateral, and in such case, the other fossa is invariably and correspondingly narrow, this suggesting at once that physical conditions may have a good deal to do with the production of the disease.

To examine this point a little further:—The initial abnormal patency of the nasal fossæ tends to the stagnation of the secretion within the nose. You know what difficulty children always experience in blowing the nose. The power of extruding accumulations of mucus in the nose is not, I think we may say, a purely natural faculty: it is an art, a trick which has to be acquired; and many mothers have great difficulty in teaching children to blow the nose satisfactorily. The difficulty consists in producing a sufficiently forcible blast of air to extrude the accumulations. Now if the nasal fossæ be abnormally wide, this difficulty will be increased. Just as a river in passing between narrow banks increases its rapidity, so the air in passing through a narrow nose increases its velocity, and becomes more able to extrude accumulated masses. If the nasal fossæ are extremely wide, the velocity of the expired air will be diminished, and the difficulty of getting rid of the accumulations increased. We have two conditions, then, favourable to the accumulation of the discharges in the nose—the width of the nasal fossæ, and the difficulty which children experience in blowing the nose.

Moreover, the disease, as far as I have had opportunities of watching it, invariably begins in an ordinary attack of cold in the head, which has a tendency to become chronic, and in which the

discharge tends to become muco-purulent rather than purely mucous. I am sure most of us have experienced the fact that towards the end of an ordinary cold in the head the mucus has become more tenacious, more yellow, more purulent. In children, especially in strumous children, this tendency, I believe, is considerable; and, of course, in strumous children there is a greater tendency for the disease to become chronic; so that we have a chronic condition of muco-purulent discharge from the nose, which tends to stagnate from the causes I have mentioned. In consequence of its stagnation it tends to dry up and putrefy, the tendency to putrefaction, I conclude, being greater from the presence of pus than if it were simple mucus. When that condition is arrived at, when the semi-inspissated mucus begins to putrefy and give fœtor to the breath, we begin to have all the conditions which we describe as *ozæna*, or atrophic rhinitis.

In this first stage of the disease there is more or less vascular engorgement of the erectile tissue, which, as I insisted at a former lecture, must not be mistaken for hypertrophy. But in the second stage, the shrinking and shrivelling up begins to appear, though at first only in the form of collapse of the erectile tissue. This, I believe, is due very much to the dessication of the muco-purulent discharge lying on the erectile tissue of the lower spongy body, and, by the contraction of the mass, it exerts sufficient pressure to force the blood out of the venous sinuses. Very little is required to effect this; the least pressure on the swollen body will temporarily force the blood away; and we may readily imagine that if a quantity of contracting mucus, which we may liken to a contracting film of collodion, is spread over this erectile tissue, it will produce a more or less permanent collapse of that tissue. As a result of this, and, I think, of the consequent *anæmia*, we have the beginning of the atrophy. It seems quite conceivable to me that these structures, being deprived of their blood by the pressure of these accumulated masses of dried up secretion, may actually begin to atrophy and shrivel up from want of simple nutrition. That, of course, gives no explanation of the process of absorption. What it is I do not know; but the fact remains that the tissues are all absorbed—absorbed, of course, by the blood-vessels. Let me repeat that it seems likely enough to me that the local *anæmia* produced by the local condition I have mentioned

may lead to this atrophy. Moreover, these patients are naturally anæmic; and in people who are anæmic, we always find more or less collapse of the erectile tissue; so that we have that factor also favourable to the continuance of the process.

From this discussion of the etiology and pathology of the condition we are led to the consideration of the symptoms; and from what I have already said you will be able to determine what some of them are. In the first place, the most pronounced symptom is that the patient wherever she goes is accompanied by a most terrible stench,—sometimes so intense that as soon as she comes into a room the odour seems to pervade every part of it. One fortunate circumstance, however, is that the patients, for the most part, and always in the later stages, are quite unconscious of it themselves, the olfactory sense being completely destroyed. It is from the friends more than the patients themselves that the complaints on this score come. This is a point of rather striking contrast to what obtains in some other affections of the nose where a fœtid smell is present, as, for instance, in the retention of pus in any of the accessory nasal cavities, *e.g.*, the maxillary, the sphenoidal or frontal sinuses. In such cases the sense of smell is not interfered with, and the patients suffer far more than the friends from the bad odour. So, too, the stench accompanying necrosed bone in syphilitic ozæna, or due to the presence of a rhinolith is, except in rare cases, clearly perceptible to the patient himself.

But to return to our atrophic rhinitis. As a consequence then of the retention of the discharge in the nose, putrefaction occurs, giving rise to a peculiar fœtor, a fœtor which is quite pathognomonic, and quite unlikely to be due to any other disease. Various theories have been started to account for it. Peculiar bacilli have been described by some and denied by others. These we need not discuss. It is sufficient to remember that the fœtor of putrefaction in different regions may be variously influenced by the association of the normal secretions. Thus in otorrhœa the stench is quite peculiar, and quite different from that of ozæna. In that case there is the cerumen, which no doubt takes part in the putrefactive process and adds its peculiar odour. So in the nose the putrefaction in this disease, where the discharge is muco-purulent rather than purulent, is no doubt modified by the mucus, producing a

totally different odour from that of retained pus in any of the accessory cavities of the nose.

The retention also leads to inspissation and steady accumulation of dried up masses in the nose, so that the nose may not be blown for weeks together. One patient assured me she blew her nose quite regularly and systematically, but only at intervals of six weeks, and then was astonished at the large amount which came away, stinking in odour, the masses being sometimes quite solid and brittle! The presence of such a putrefying foreign body in the nose tends to make matters worse, increasing the mischief in the mucous membrane, increasing the amount of muco-purulent discharge, and often leading to suppuration in the accessory cavities of the nose, and sometimes even to caries of the middle turbinated and ethmoidal bones. Yet it is only very exceptionally that we find suppuration in one or other antrum in the frontal sinus, and so on. Nevertheless, we must always be prepared to find some real suppuration in addition to the muco-purulent discharge. These cases are often very complicated. The affection of itself, when not complicated, is simply a disease of the mucous membrane. There is no ulceration and no caries; it is simply a muco-purulent discharge produced by a chronically inflamed mucous membrane.

Of course, the inspired air in its passage through the nose does not become moistened as it should, and reaches the pharynx and larynx in a dry state; consequently, the pharynx of these patients has its posterior wall dry and glazed, although not necessarily covered with a muco-purulent secretion, being generally glazed with a simple dry mucous film. This alone causes great discomfort in the throat; and it is often for this condition that the patient primarily seeks advice, thinking the condition of the nose a small matter. Of course, we can do nothing for the throat in such a case until the nose is put right. The tendency to dryness exists in the larynx too, so that frequently large masses of dried up mucus are found there which by their very presence induce a chronic laryngitis and various degrees of dysphonia. Extraordinary degrees of chronic laryngitis may be produced simply by the inability on the part of the nose to moisten the inspired air before it reaches the larynx. One patient of mine, a lady who was over 40 when she came to me, had been suffering nearly all her life from this affection; and her great trouble at the time was that she was growing, as she con-

sidered it, asthmatical. On inquiry and examination, however, one discovered that her difficulty in breathing was due to the accumulation of vast masses of mucus in the trachea itself—more in the trachea than the larynx in her case. This was due primarily, I believe, to the dryness of the nasal mucous membrane. The constant irritation of the dried masses in the larynx and trachea, no doubt tended to perpetuate the condition. She would cough out large masses of this semi-inspissated mucus, which formed as perfect casts of the trachea as we sometimes find in diphtheria, after the extrusion of which she had great relief. That was the worst case of the dryness extending downwards I have ever seen.

Apart from the accumulation of the mucus in the trachea we may get real asthmatic symptoms produced, just as in cases of nasal obstruction; because in such a case as this, although the nose is abnormally patent it can no longer perform its functions; and, as a matter of fact, the patient is probably better when breathing through the mouth than through the nose. So that many of the consequences of buccal respiration may be present.

As I have said the patients are always anæmic, and the disease tends to produce anæmia. As soon as the patients mend in the nose they improve in general health to a remarkable degree.

One satisfactory point about the disease is that it tends to improve as the patient grows older, as is clearly shown from the common observation that these patients seldom present themselves for treatment after thirty-five years of age. I have seen very few patients as old as that. Since there is no real reason why a patient should not be as willing to be treated for it when older as when younger, we necessarily assume the disease has a tendency to spontaneous amelioration. When we do, moreover, see patients as old as thirty-five the symptoms have become modified considerably, the fœtor, for instance, being less marked. Why it should tend towards spontaneous improvement I do not know, as the physical conditions remain unaltered; the atrophy can never be cured, and the nasal fossæ remain abnormally patent. Still you may hold out the hope that even if not treated, or if treatment is not so successful as it ought to be, the disease will, in the course of years, tend towards spontaneous amelioration.

As to diagnosis there is not much difficulty. But a good deal of confusion has arisen between ozæna and two or three other affections on

account of the fact that this atrophic rhinitis is not the only disease in which we get a dryness of the mucous membrane; and the question has been raised by some specialists as to why some cases of "dry rhinitis," as they like to call it (*rhinitis sicca*), should be associated with a bad smell, and others not. The reason is simply that the affections are entirely different. I think we may say there are three conditions in which there is present a dryness of the mucous membrane. The first and mildest of these is simple anæmia of the mucous membrane of the nose, where we have collapse of the erectile tissue, insufficient supply of blood to the mucous glands of the inferior turbinated body, and consequent diminution of the secretion. These patients present a certain amount of dryness of the mucous membrane, which is especially seen over the anterior extremities of the middle turbinate bodies. But the dryness never leads to the accumulation of large quantities of mucus, although a little inspissated film may be seen. This condition is not a disease, but simply a symptom of general anæmia. I believe every anæmic person has more or less collapse of the erectile tissue of the nose. It nevertheless calls for treatment, for this reason, that it is sometimes accompanied by a dryness of the throat and larynx, and it is for this that the patient seeks relief.

I remember an interesting case of this sort. A patient came to me complaining of difficulty in singing. She had a nice singing voice which of late had failed. This she could not account for, as she believed herself in good health. She presented the ordinary symptoms of a slight chronic laryngitis. On examination further one noticed that the posterior wall of the pharynx was a little dry and glazed. This suggested an examination of the nose. The inferior turbinated bodies were found to be collapsed, and the whole interior of the nose slightly dry. This was sufficient to account for the trouble in the larynx. The patient at first sight did not appear to me to be anæmic. On further examination, however, we found symptoms of anæmia, although the cheeks and lips were of good colour. The patient was treated in all sorts of ways, but these failed altogether to do her any good. She had tonics of every kind, and Iron in every form; she was sent to the seaside to be braced up, but without benefit. One day, however, when I had practically given up treatment of her case, she came to me and said, "I think you will find me better." I examined her larynx,

and saw the condition was practically cured. Her turbinated bodies were properly turgid, her nose and the posterior wall of the pharynx moist. I was rather surprised, and asked what she had been doing. "Nothing at all," she said, "my voice has got better of itself." I was very much pleased, and told her she would require no further treatment. Just as she was leaving, however, she incidentally remarked that within the last three months she had become engaged to be married! And that, Gentlemen, was the explanation of the whole improvement in her condition and in the quality of her blood, and I suppose of the improvement in the inferior turbinated bodies. The erectile tissue had swelled out, the mucous membrane had begun to secrete, and her vocal cords returned to their normal condition. The story is worth telling as an instance of the importance of attending to all sorts of remote general conditions, as well as to the near local conditions that present themselves to the specialist. One might even cite the case as an instance of the dangers of specialism! One might have punished that poor girl's nose with all sorts of applications without doing any good, whereas if one could have induced her to get engaged to be married no further treatment would have been necessary!

(*To be concluded.*)

THERAPEUTICAL NOTES.

Variocoele.—Dr. Sebileau usefully divides variocoeles into three classes, as follows:—

Class I.—Those on which an operation *must* be performed. The class includes: 1. Those which are very painful. 2. Those causing testicular atrophy, with interference in the generative faculty. 3. Those causing obscuration of the mental faculties indirectly or directly. 4. Those which oppose a serious obstacle to the due performance of the daily work of the patient. In any of these four groups the size is immaterial.

Class II.—Those on which a surgeon *may* operate. This class includes: 1. Those which are very large, but occasion no great inconvenience. 2. Those which, while neither painful nor very large, cause the patient to solicit a radical cure.

Class III.—Includes all other forms of variocoele. The treatment here *must* be palliative only until one of the circumstances belonging to Class I. or II. arises.—(*Gaz. Med. de Paris.*)

Facial Erysipelas.—Azotate of Crystallized Aconitine, in doses of one fiftieth of a milligramme, will often lessen the duration and relieve the pain of this distressing malady.

(*New York Med. Rec.*)

REVIEWS.

Injuries and Diseases of the Jaws. By CHRISTOPHER HEATH. Edited by H. P. DEAN. 4th Edition. (J. & A. Churchill.)
Published Price 14s.

By the time a monograph has reached its fourth edition detailed criticism is unnecessary, for the fact itself speaks for the general excellence of the work, and shows that it is appreciated by a large number of readers. Mr. Dean has brought the pathology up to date, though we are sorry to see the latest disease, viz., acromegaly, omitted from the index, although it is mentioned in the body of the work under general hyperostosis. As a book of reference in any case of injury or disease of the jaws we can confidently recommend the book, because of the large proportion of space which is devoted to treatment, which is after all the primary object of our profession.

The Rectum and Anus: their Diseases and Treatment. By CHAS. B. BALL. 2nd Edition. Cassell's Clinical Manuals.

Published Price 9s.

This is another monograph of very great interest and practical use to members of the profession, to whom a consultation is a matter of difficulty. As a test of this, it is natural to turn to a common affection, e.g., piles, and here the information given is at once most practical and yet complete and satisfactory. Other chapters on rarer affections of the anus we have found to stand the test equally well, and we congratulate Mr. Ball on the demand for a second edition of his most excellent manual.

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THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 28, 1894.

A LECTURE ON BRONCHITIS.

Delivered at Charing Cross Hospital, Jan. 29, 1894, by

T. HENRY GREEN, M.D., F.R.C.P.,

Senior Physician to the Hospital;
Physician to the Hospital for Consumption, Brompton.

GENTLEMEN,—Our subject to-day is bronchitis. The consideration of bronchitis at this season of the year is obviously appropriate, as the disease abounds both in the wards and the out-patient room. You may say that it is so common and you are so familiar with it, that it is hardly worth while to make it the subject of a special lecture. But you must remember that in its acute forms it is an extremely fatal disease; that it is especially prone to be chronic and intractable, and thus to lead to secondary changes in the lungs and bronchi, which enhance and modify its symptoms; that there is great diversity both in the bronchitis and in the patient—it may be idiopathic, or symptomatic of a disease more important than itself; and lastly, that its successful treatment involves a careful consideration of all the etiological factors in the case before you, an accurate estimate of the extent and severity of the bronchial inflammation, and of any resulting alteration in the lung tissue—in other words, a comprehensive diagnosis.

The subject is a wide one, and we must restrict ourselves to-day mainly to the more chronic forms of the disease. Acute and dangerous bronchitis occupied us on a previous occasion. I do not pretend to have anything new to tell you. My object will be to emphasize what a somewhat large experience has taught me to think important.

DIAGNOSIS.

The *recognition* of the bronchitis rarely presents any difficulty. It should be based on physical signs rather than on symptoms. I need hardly caution you against the mistake of concluding too hurriedly that cough, even when attended with expectoration, is bronchial in its origin. You will remember how frequently this is due to the pharynx. In the absence of those adventitious sounds, dry and moist rhonchi, with which you are all familiar, you are not justified in diagnosing the existence of

bronchitis. The only exception to this is where the inflammation is confined to the trachea and largest tubes (tracheal catarrh). In such cases no rhonchi may be audible, and symptoms alone must guide us. But it is rare for the process to be so limited as not to give rise to an occasional wheeze.

With regard to physical signs—they may want carefully looking for. They come and go, audible at one moment they may be imperceptible the next, and frequently they are developed only by cough. Examine the whole chest, and don't forget the posterior bases.

I must remind you briefly of the mechanism by which the adventitious sounds are produced, as this is of importance in diagnosis. The *dry* rhonchi—sonorous and sibilus—usually more audible with expiration than with inspiration, are due to narrowing of the tubes by adherent mucus, swelling of the membrane, fibrous thickening, or spasm. When due to mucus, they can often be dispelled by cough. Swelling of the membrane obstructs especially the smaller tubes, and gives rise to sibilus. Fibrous thickening may give rise to more or less permanent rhonchus. Do not forget this.

The *moist* rhonchi indicate for the most part implication of the smaller tubes, the finer ones—almost exclusively inspiratory—tubes of smallest calibre, such as we get in capillary bronchitis. The larger moist rhonchi, usually both inspiratory and expiratory, indicate usually abundant secretion and its accumulation from incomplete expectoration. They may be met with in cases of moderate severity, and should be looked for at the posterior bases.

Having thus recognized the existence of bronchitis, and by a careful examination of the chest estimated its extent and severity, we proceed to complete our diagnosis. Remember that bronchitis is exceedingly liable to be associated with other diseases which stand in more or less causal relation to it. Is, then, the bronchitis idiopathic, or is it symptomatic of some other disease, such as one of the acute exanthemata, cardiac, or renal disease, or pulmonary tuberculosis? To search for any etiological factor in addition to the climatic one—which doubtless occupies a prominent position in most cases, and may be the sole cause of the

disease—constitutes a most important part of the diagnostic problem. It is one we are too apt to overlook, hence many of our errors in prognosis, and failures in treatment.

Let us consider briefly this association of bronchitis with other diseases in so far as it affects the question of diagnosis under the following heads:—

1. THE ACUTE SPECIFIC DISEASES.—The only association of bronchitis with the acute specific diseases which might possibly lead to error in diagnosis, is that with typhoid fever. In mild cases of typhoid where slight pyrexia and some sonorous and sibilus over the chest may be the only objective symptoms, it is possible to conclude that your patient is suffering from a simple catarrh and to overlook the existence of the typhoid fever. I have seen more than one such mistake; and on one occasion it led to disastrous consequences. I would ask you in this connection to remember that *physical signs of bronchitis, plus pyrexia, is always suspicious*. Simple bronchitis does not usually cause pyrexia, except perhaps during the first day or two of the illness. The existence of pyrexia should always lead you to search for something *in addition to the bronchitis*—it may be one of the acute exanthemata, it may be pulmonary tuberculosis, it may be broncho-pneumonia. Remember this, and with a little care you will not be likely to fall into error.

2. PULMONARY TUBERCULOSIS.—The association of bronchitis with pulmonary tuberculosis is much more important, and is frequently the cause of real difficulty in diagnosis. Cases in which an examination of the chest yields nothing unequivocal but physical signs of bronchitis, and yet in which tubercle is the important lesion are common. I am speaking, of course, of a more or less general bronchitis, not of that inflammation of the smaller tubes limited to the upper lobe of one lung, which give rise to the physical signs met with in such a large number of cases of early phthisis. The localization of the bronchitis in such cases is almost conclusive evidence of its tubercular nature. To discuss this question at all fully would involve a consideration of the diagnosis of pulmonary tuberculosis in its more unusual manifestations. I must content myself now with reminding you—(1) that in the ordinary cases of phthisis, where the disease is limited to the upper lobe of one lung, the supervention of a general bronchitis may completely mask the physical signs of the apex lesion; (2) that

simple bronchitis may be the initial lesion preceding the development of a localized or more or less general pulmonary tuberculosis; and (3) that certain cases of pulmonary tuberculosis which are characterized by miliary and widely disseminated lesions, which tend to become fibroid rather than to disintegrate, are very frequently associated with so much bronchitis and emphysema that their tuberculous nature is altogether overlooked.

The diagnosis of these cases must be based on an examination of the sputum for the bacillus of tubercle. Remember that, although in the great majority of cases of phthisis an examination of the sputum is quite unnecessary for diagnosis, in cases in which the tuberculosis is masked by bronchitis, emphysema, pleurisy, etc., an examination of the sputum may be the only means of arriving at a conclusion. The discovery of the bacillus settles the question; but be very careful how you draw conclusions from a *negative* result. The sputum should be examined again and again, and even then the evidence is by no means conclusive. The diagnostic significance of pyrexia I have already alluded to. It points to tuberculosis and to the necessity of searching for the bacillus. The absence of fever, on the other hand, by no means excludes tuberculosis: the cases of disseminated fibroid tubercle associated with bronchitis and emphysema, are often non-febrile.

3. HEART DISEASE.—The very frequent association of bronchitis with disease of the mitral valve, is only likely to lead to difficulty in diagnosis in those cases in which the valve lesion is latent, and is unrevealed by unequivocal auscultatory signs. In such cases, although the bronchitis is obvious, the mitral disease too often escapes detection. The diagnostic significance of large right heart under these circumstances I have often insisted upon. You will remember, of course, that the liability to bronchitis in mitral disease is due to the interference with the pulmonary circulation caused by the valve lesion, and that this interference, in marked contrast to the interference with the systemic circulation, exists quite independently of failing compensation.

4. CHRONIC GENERAL DISEASES AND DIATHETIC CONDITIONS.—The frequency with which chronic Bright's disease, gout, rickets, syphilis, plethora, etc., constitute prominent etiological factors in bronchitis, it is all-important to bear in mind. Such diseases not only favour the occurrence of the bronchitis, but they tend to increase its per-

sistency and its liability to relapse and recur. A careful search for such causes should always be included in our diagnosis. Forgetfulness of this is responsible for many of our disappointments in therapeutics.

PATHOLOGICAL CONSEQUENCES.

There are certain pathological consequences of bronchitis which it is important to bear in mind.

Liability to recur. Each attack of bronchitis leaves an increased vulnerability of the mucous membrane behind it, and a consequent liability to relapse and recurrence, the disease thus tending to become chronic. Hence the paramount importance of doing everything in our power to re-establish the health, especially in first attacks, and where there is any inherited or acquired tubercular tendency.

Of the pathological changes induced by the bronchitis in its acute and chronic forms, pulmonary collapse, broncho-pneumonia, bronchiectasis, bronchial thickening and fibrosis, and emphysema—it is only of the two last that I shall say a few words.

More or less *thickening of the smaller bronchi*, involving the mucous membrane, the muscular and fibrous structures, and often leading to irregular narrowing and dilatation of the tubes, is met with in most cases where the disease is markedly chronic. Such changes must obviously favour the persistence of the catarrhal process, and tend to interfere with expectoration. They also account for that prominence of physical signs which are so often out of all proportion to symptoms. Remember this, and you will understand the abundant sonorous rhonchi and sibilus met with in so many cases, and persisting for long periods, unaccompanied by cough, expectoration or other symptoms of bronchial inflammation.

Emphysema is such a frequent consequence of chronic bronchitis, and has such an important bearing on its symptomatology and treatment, that although its consideration must be reserved for a future occasion, some points connected with it must be here alluded to. Remember, in the first place, that emphysema favours the occurrence of bronchitis, whether itself the outcome of the bronchial affection or owing its origin to other causes. Given emphysema, the liability to bronchitis is materially increased, and some persistency and obstinacy of the disease are to be anticipated. The emphysema obviously must also increase the

gravity of the bronchitis. The interference with respiration due to the emphysema is added to that caused by the bronchitis, and may greatly increase its danger. But, in addition, the emphysema obstructs the pulmonary circulation, and thus tends to cause venous plethora and those nutritional and structural changes in the stomach and liver, and consequent dyspeptic troubles which result from this disturbed circulation, and with which you are all familiar. True, in most cases, this interference with the systemic circulation is counteracted by compensatory hypertrophy of the right ventricle; but the tendency is, nevertheless, a more or less continuous one, and one which it is most important to bear in mind in the treatment of our patients.

TREATMENT.

In the treatment of bronchitis, we must consider whether the disease is acute or chronic, recent or old, the size of the tubes implicated, the evidence of accumulation of the secretion, and the etiological factors in the case before us, especially the existence of those chronic diseases to which we have already alluded. Remember the paramount importance, especially in chronic and obstinate cases, of a comprehensive diagnosis.

The therapeutics of bronchitis is admittedly unsatisfactory. Our knowledge of expectorants is very incomplete, and each is apt to advocate his favourite drug. There is no royal road to success; but guided by sound pathology and clinical experience, we may anticipate satisfactory results. In the brief time at our disposal, I cannot do more than indicate some of the more important points.

First, in *acute* and recent cases, remember the great importance of endeavouring to arrest the disease and prevent its extension to the smaller tubes. Be prompt in your treatment. Confinement to the house, a warm moist atmosphere, a mild purge, some saline mixture with Ipecacuanha given warm, and warm and moist applications to the chest are the most important. At the onset of the disease Ipecacuanha is the most useful drug. Do not be afraid of it—ten or fifteen minim doses every two hours for the first day or two of the illness, or until the expectoration is free and the skin perspiring. Tartar Emetic might be used much more frequently than it is with distinct advantage. Apomorphia is a useful drug. Hot moist applications to the chest are all important. They are better than liniments in the early stage

of the disease, and, provided you can have an efficient nurse, nothing answers so well as a good jacket-poultice.

With the subsidence of the more acute symptoms diminish the dose of the Ipecacuanha, and, as soon as you can, discontinue it. Substitute some stimulating liniment for the poultice. We are apt to err by continuing our expectorant remedies too long, and postponing stimulants and tonics. Some Nux Vomica with Ammonia and Bark, with the addition, if necessary, of a little Ipecacuanha and Tinct. Camph. Co., is most useful at this period; and later, some acid with Quinine or Strychnine. Remember the liability to relapse and recurrence, and do everything in your power to promote the complete re-establishment of health. Impress on your patient the necessity for careful supervision. In many cases change of climate—a fortnight at the seaside—is necessary to effect this result. Any inherited or acquired tendency to tuberculosis, of course, enhances the importance of such precautionary measures.

In the treatment of *chronic* cases you will be wise to anticipate trouble. Look at the case outside the bronchitis and see if you can discover any etiological factor, such as gout, renal disease, or plethora, which you can influence. Too often climate appears to be the only cause, and from the circumstances of the patient, you have no power to alter it. In the management of these cases I would say, speaking generally, put the general health first and the lungs second. If you take care of the general health the lungs will often take care of themselves. Withhold expectorants and lung remedies unless the super-vention of acute attacks or of some special symptoms seems to call for them. The continuous and indiscriminate drugging with cough medicines, so common in the treatment of such cases, is, I think, distinctly harmful.

In attempting to improve the general health, let the stomach and digestion receive your first attention. Some vegetable bitter, with Sodium Bicarbonate, given two or three times a day about ten minutes before food, is usually markedly beneficial. Nux Vomica and Carbonate of Ammonia may be added to this mixture, and will be useful in aiding expectoration, and also, if necessary, a few drops of Ipecacuanha Wine. An occasional dose of Blue Pill and Colocynth to obviate the tendency to venous plethora must not be forgotten. This is especially important

when the bronchitis is associated with emphysema. Cod Liver Oil and Arsenic are often most beneficial taken after food.

Counter-irritation is of special value. Some stimulating liniment should be rubbed thoroughly into the chest night and morning. What I generally use is the Turpentine and Acetic Acid Liniment of the Pharmacopœia. The benefit is usually most marked when there is emphysema, and in these cases a Croton Oil Liniment is often more useful. Patients will willingly submit to the discomfort of extensive pustulation of their chest in order to secure the relief which it gives to their symptoms.

If, in spite of such treatment as I have indicated, the cough and expectoration persist, you may have recourse to special remedies—Tar, Turpentine, Terebene, Benzoin, etc.; also the inhalation of such substances in the form of vapour or spray. Give these a trial. What answers in one case may not in another. I do not think I can give you any useful rules for your guidance.

In the treatment of special symptoms—when the sputum is copious and puriform, mineral acids, with Strychnine or Quinine, are often of great service—more so than the gum resins.

Paroxysmal dyspnoea, asthmatic symptoms, which form such a prominent feature in many cases, are usually best met by Iodide of Potassium and Stramonium. Do not forget the important rôle played by the stomach in many of these asthmatic cases. Restricting the last meal, washing out the stomach by a drink of hot water before going to sleep, and regulating the bowels, will often do much to diminish the asthmatic attacks.

Climatic treatment, the only means in too many cases of rendering life endurable, we cannot now discuss.

Tuberculosis of Lungs.—Continuous inhalations of Peppermint Oil, internal administration of an alcoholic solution of Creasote, Glycerin, and Chloroform, and one per cent. of Peppermint Oil. Thirty-nine cases were thus treated with excellent results, the lungs being apparently healed. Bacilli disappeared in from two weeks to two months, the bodily weight increased, the appetite improved, the cough, sweating, and expectoration disappeared.
(G. M. CARASSO, *Gazzetta degli Ospitali*.)

A CLINICAL LECTURE

ON

CARIES.

Delivered in connection with the London Post-Graduate Course in the Hospital for Sick Children, Great Ormond Street, Jan. 25, 1894,

By **JOHN H. MORGAN, F.R.C.S.,**

Surgeon to the Hospital.

GENTLEMEN,—I am sorry to say that I must begin with an apology. When I was asked four months ago the subject for lecture at this time, I naturally gave one which I thought I could illustrate from cases that I could accumulate and collect beforehand; but, unfortunately, owing to unforeseen circumstances such as we can never anticipate, my ward during the last fortnight had to be closed owing to an outbreak first of diphtheria and then of scarlet fever. One child was lost through diphtheria, and another was very ill with scarlet fever, and had to be removed from the ward; then a nurse was taken ill with scarlet fever, and I had to act in a summary manner, and close the ward. Having had these epidemics in my own ward too, I cannot borrow from the wards of my colleagues; furthermore, in the ward of one of them there is another outbreak of diphtheria, which has caused him to empty his beds. All these circumstances prevent me from illustrating the subject, as I had hoped, by the exhibition of patients. I have, however, managed to collect a few cases, which I will afterwards show you.

The specimens that I can put before you, again, are very few. Those which illustrate caries in its earlier conditions are necessarily rare—the cases in which it leads to operation or is found during operation being generally far advanced.

The subject is one of great interest, since the disease is one that fills, I believe, one-third, if not one-half, of the beds in a surgical ward of such a hospital as this. Caries in some form or another is the commencement of so many serious conditions of disease that that estimate is, I believe, not an exaggeration.

The early condition of caries, as I have said, is hard to exemplify. The only opportunity one gets of doing so is where a patient dies from some intercurrent disease, and in whom conditions of early caries are found, the caries being either, as it were, secondary to the same disease existing in

some other part of the body, or arising in some unsuspected region where the patient has died from some fever, or possibly some other manifestation of tubercular disease.

Again, caries is a term to which we may give a much more definite application now than those of us who studied pathology, say, twenty years ago, could do. We were, as I think all will bear me out in saying, dumfounded by the many different forms of disease which were covered by this word. Now, however, owing to the progress of pathology, caries is a term with a much more definite limitation. We may, I think, describe it as a definite septic disease of bone, due to the localization of irritant microbes, carried by the blood-vessels or lymphatics. It is in those bones where growth is most active, and therefore where vascularity is greatest, that caries is most frequent. Consequently, we find it occurring in various bones in different proportions and at different ages, according to the extent of physiological change occurring in each part. That, I think, is a fair definition of the term caries, which formerly, as you know, covered all the different forms of ulceration of bone, whether these were due to injury, to syphilis, or to this disease which now is traced to its proper source, namely, the presence of a bacillus.

In considering this subject, it is very important to try and analyze to what extent this disease is due to injuries. We are always taught to inquire in a case of caries how far it is due to injury; and we invariably do so: but I cannot help thinking that the extent to which injury is responsible for the development of caries is very often exaggerated. We find a case that we recognize as caries, and we at once ask whether the child had a blow or a fall. We are told, perhaps, that the child did have a fall; but most children have them pretty frequently. Whether the fall had any definite relation to the outbreak of disease is seldom fully investigated; and I am inclined to think that, as a rule, we attribute far too much to injury as the cause of the outbreak of this disease. That it frequently follows slight injury in predisposed persons cannot, of course, be denied; but there is too often a tendency to seek for a history of injury to account for the presence of disease. Hence, many cases of caries are ascribed to injury which, in all probability, are due to a natural course of disease.

Then comes the subject of scrofula; and in this diathesis caries, of course, develops as a rarefying osteitis, and as a result of the proneness of such

persons to sluggish inflammation, not only of bone, but of other tissues. That this is so, we see by the frequent development of caries where there exists some other manifestation of the tubercular diathesis.

Let us now briefly consider the changes that take place when caries commences. At first there is a general increase of vascularity which is hard to distinguish from the normal condition of the vascular medulla. Then the medullary tissue becomes changed into masses of granulations, the cancelli break into one another and gradually disappear. To the further changes which ensue various names have been given, which indicate different developments of the process. If these granulations become profuse and generate pus they invariably make their way to the surface; the periosteum becomes raised and inflamed, the pus bursts through the overlying tissues, and we have a condition that is known by the term caries fungosa, or, in English, fungating caries. If the granulations undergo fatty degeneration they form a lowly organized material which goes by the name of caries sicca, that is to say, a mass of caseous material surrounded by granulations, from which there is no definite discharge of pus, which lies often quiescent mixed with the granular debris of the bone, and in course of time undergoes changes of a retrograde or of a developmental character, either breaking down and becoming a mass of pus, or being organized so as to form fibrous tissue and eventually bone, or, if the process of disintegration is so rapid that a large proportion of bone is destroyed *en masse* the condition of caries necrotica is produced.

If, then, we regard caries as thus defined, we may put aside all those other forms of ulceration of bone, whether caused by injury or due to the results of syphilis, either in adults or young people.

Another difference between caries as thus defined and syphilitic ulceration of bone, as exemplified in the case of children, is that caries very seldom attacks the flat bones, syphilis more frequently than not. Some years ago, when I was working in the out-patient room here, I tried to ascertain the various proportions in which tubercular disease attacks the various parts of the body, the joints particularly, I made a careful analysis of all the cases that came under my notice during the years 1879-80. That analysis is curious. Of course I would not be justified in

saying that all these cases are cases of caries, but they were cases of disease of the various joints of the upper and lower extremity, and no doubt a very large number of them were due to caries of the epiphyses, and not entirely due to disease of the synovial membrane. The results came out in this way. The total number of cases that passed under my observation were 2869 in these two years. In the upper extremity seventeen cases of joint disease were noticed,—five at the shoulder joint, nine at the elbow, and three at the wrist. On the contrary, in the lower extremity there were 141 cases of disease of the joints,—ninety of these occurred in the hip, forty in the knee, and eleven in the ankle. These are statistics of considerable interest, and I am not aware that a similar analysis is to be found in any of the ordinary works on surgery. Taking these figures as representing fairly the proportionate frequency of tubercular disease in the various joints, we must subdivide the various parts of these joints; and, seeing how very much larger the proportion of disease is in the lower extremity than in the upper, it will be well to begin by examining the cases of disease of the joints of the lower extremity before going to the upper.

Taking, first, the ankle and tarsal bones, I think we shall be correct in saying that the os calcis is more frequently diseased than any other of the bones of the foot. Whether that be due to its liability to injury or to the very large amount of cancellous tissue of which it is composed it is again rather difficult to say. The disease is limited to this bone for a much longer period than it is to any other bone of the tarsus, partly because the bone is larger, and partly because its articular surfaces are not so extensive as in the case of the astragalus and cuboid. Disease once commenced in the os calcis, therefore, remains confined to that bone for a much longer period than to any other bone in the foot. If we take it as an example of the progress of caries we may enumerate the symptoms we should observe in such a case chiefly, as (1) early tenderness and pain, these, perhaps, appearing before the lameness which, sooner or later, inevitably follows. Then comes (2) swelling, due to the process that I have just described. Usually this is to be seen on the outer, but very often on both sides. If this is not arrested by rest the swelling increases, and (3) an abscess forms, granulations, typically puffy, with slight serous discharge ap-

pearing on the surface, and a probe passed through this mass of granulations finds a gritty condition of the bone sometimes extending to the opposite side.

Coming next to the astragalus—this is seldom involved without implication of the ankle-joint. It is generally to be recognized by swelling and puffiness at the ankle, with an atrophied condition of the parts beyond. That well-known puffy appearance of the ankle with the veins over it distended, and the very atrophied condition of the parts beyond, is almost typical of the disease of the astragalus. When this condition has existed, we naturally look for some further development, and examine, either with or without an anæsthetic, for grating, such as we would expect where so large a surface of bone is denuded of cartilage. But we must not conclude that we have made a mistake if we find that the grating does not exist. This is a point to be remembered, too, in connection with almost all the other joints in which strumous or tubercular caries is likely to occur. Perhaps it is most frequently the source of error in cases of affection of the hip. In that joint, where caries no doubt very frequently is the origin of the disease, on examining without an anæsthetic and even with one, it is not by any means unfrequent to get a considerable amount of movement of the head of the bone in the acetabulum, without detecting anything like grating of the two surfaces together. This, of course, is due to the fact that both surfaces, though denuded of cartilage, are covered by a thick layer of granulation tissue. And so it is with regard to the astragalus, which may be in a far advanced state of disease, but so covered with granulation tissue, that free movement under an anæsthetic does not give rise to any feeling of grating in the parts. A little boy whom I shall show you in a few minutes was in exactly this condition. He has disease of both ankle-joints commencing undoubtedly in the astragalus. A few days ago his right ankle-joint, which had been protected by plaster of Paris for many weeks, appeared to be giving rise to more pain. The plaster of Paris was, therefore, removed. I put him under an anæsthetic, and examined the ankle-joint. Although it was obvious that the disease was extensive, there was no grating whatever when the two surfaces were rubbed on one another. In that case I made a free incision and removed the whole of the astragalus, which we found to be absolutely denuded of all cartilage, and covered with granula-

tions, as were also the surfaces upon which it ought to have moved.

Passing briefly to the other bones of the foot, we find that this form of disease is seldom confined to the scaphoid, because it generally involves through the synovial membrane, which is so profuse in that part of the foot, the cuneiform and astragalus.

The cuboid bone, again, is seldom primarily involved, probably owing to its earlier ossification—at least, this is the explanation suggested by Mr. Jacobson—the earlier ossification of the bones on the outer side of the foot.

The metatarsals are very frequently the seat of carious disease in children, and luckily they do not necessarily involve the implication of other bones of the foot so frequently as do the bones of the tarsus. But, as I shall show you again in the ward, whenever the metatarsals are diseased, the fear is that the affection will spread either to the epiphysis of the bone itself, and so to the bone with which it articulates, or that what arises in one metatarsal will arise spontaneously and separately in the tarsal bones. Whatever be the cause, the danger that one always has to fear whenever caries exists in a metatarsal bone, is that it will spread to the other bones of the foot; and in a great many cases unfortunately it does. My experience is that in few cases in children is the disease confined to the metatarsals, however early one takes it in hand. It is always, therefore, to be regarded as more or less serious on account of its probable implication of other parts.

Coming to the larger joints at the lower extremity, I may touch upon the fact that this form of disease is so frequently found to implicate the epiphyses of the bones of the lower limb,—far more often than it does those of the bones of the upper extremity; and that will bear out the statistics I have just alluded to. But again the verification of this fact is often difficult because disease which may have commenced in an epiphysis or at the epiphyseal line spreads so rapidly to the synovial membrane that perhaps this tissue shows the more prominent signs of disease, and the original surface from which it started is not recognized until erosion or amputation is performed, and the results of the destruction of bone are shown to be in advance of the changes in the synovial membrane. Certain it is that in a large majority of the amputations and excisions of the knee and of the hip-joint that are done, we have reason to think the disease com-

menced first in the epiphysis, and spread to the synovial membrane.

Of the epiphyses of the lower extremity those most frequently involved are undoubtedly the upper epiphyses of the tibia, then the epiphyses of the lower end of the femur, and then the epiphyseal head of the femur which gives rise in so many instances to necrotic caries of that piece of bone, and to the so-called morbus coxæ.

My own view is that more cases of joint disease in children commence as epiphysitis,—as tubercular disease of the epiphysis,—than as disease commencing in the synovial membrane.

I shall just allude to the one other part of the body where perhaps caries is more frequent than in any other, namely, the spinal column. The examples of this are, of course, very frequent both in the out-patient room and in the wards. Here caries is much more prone to implicate the body of the bone and its anterior surface than any other part, although we were taught, and some people still believe, that the disease commences in the epiphysis that forms the lower and upper part of the body of every vertebra. From the bodies it spreads either backwards or forwards, and the matter which in many cases is formed, though not in all, makes its way according to the direction in which it is guided either by the anterior or the posterior common ligament. Most frequently it passes under the anterior common ligament and spreads to one side or other of it, and then is guided by circumstances to some other point. It is the parts surrounding the point of exit from the anterior common ligament that guide its progress and determine its point of presentation on the surface.

In the case of the spine the difficulty to which I have alluded of determining the actual spot at which the disease commences is even greater than it is in other bones which are more readily accessible. In the vertebræ we very seldom have an opportunity of seeing anything but the advanced condition of disease, whereas in bones like the os calcis, etc., we may by means of an incision note the condition at a very early stage of its progress. Occasionally, however, we do find that where the disease has existed in the spine to a considerable extent in one part, it may be seen in a very early stage at another, and thus we are able to study the earlier manifestations of this form of disease. Without going into details, we find that the disease in these bones commences and

progresses in the same way as I have described in connection with the os calcis, so that we need not dwell on its development in any of the other bones. I need not describe again the progress of the changes which come later, namely, the process which takes place in the granulations, or the course which pus takes in presenting at the surface,—that is more worthy of another lecture on the subject of spine disease, and limited to that.

I am sorry that one very interesting case of spinal disease which was under my care the other day has been sent home, but sent home, I am glad to say, very well. It was one of very considerable interest. I was asked by my colleague, Dr. Sturges, some months ago to see a child supposed to have caries of the cervical vertebræ, who had complete paralysis of the lower extremities, some interference with the power of the sphincters, and a very considerable interference with the movements of the arms and hands, all indicating a considerable amount of pressure high up in the cord. The respiration was slightly interfered with, and one's prognosis was, of course, extremely bad. The child lay in this condition for many weeks. I had ordered her a jacket to keep her perfectly quiet. In some five or six weeks I was asked to see her again on account of a swelling that had appeared on the right side of the neck, which left no room on examination for doubt that it was an abscess presenting pretty prominently above the sternum, and on the inner side of the sternomastoid. This was opened and a fair amount of the usual grumous pus was evacuated. From that time the child began gradually to improve, and,—to make a long story short,—her paralysis entirely disappeared, and she went home very shortly with a jacket applied, and with no trace either of abscess or paralysis. No doubt the abscess before it made its way forward was pressing backwards, and in some part or other by pressure on the theca of the cord was interfering with the cord itself, so as to cause paralysis; but directly it found an easier way to the surface of the body these symptoms were relieved and the paralysis disappeared; and I hear that the child is comparatively quite well.

I shall not trouble you with more remarks, as I think you will find more interest in the few cases illustrating caries that I have to show you in the ward. I very much regret that they are not more numerous, but the cause I mentioned by way of

explanation must, I think, be accepted as one that could not be avoided.

Here is a boy with double disease of the ankle-joint, from which a little over a fortnight ago I removed the whole of the right astragalus. The articular surfaces were entirely diseased, and the bone lay as a loose sequestrum between the bones with which it articulates. I took it away without any difficulty through an incision on the outer side, and scraped all the granulation tissue from the other bones. Since that he has done exceedingly well. The contour of the foot is very little different from the normal condition, but very different indeed from what it was before I operated.

The left foot has not been dealt with quite so thoroughly. Here the parts were scraped, and the case has gone on fairly well; but I am afraid that, as in the case of the right foot, we shall yet have to do something more radical. It has improved since I last saw it, when I scraped it again the other day.

It is not often that one sees double disease of the ankles, though, of course, one often sees tubercular disease manifested in more than one joint. Here, for instance, is a boy who had first disease of the left knee, entirely tubercular. The knee was first scraped, and did well; but unluckily he got some hæmorrhage into the parts, and the thing had to be dealt with by opening and scraping again. Then it went on badly, and finally amputation had to be performed, so that he was left with only one leg. Unfortunately, whilst he was still in the ward disease developed in the ankle-joint of the remaining limb, and that, of course, had to be dealt with in some way or other. I was very loth indeed to do anything very radical; I ought to have amputated, no doubt; but seeing the little fellow had only one limb, I hesitated to do that, and besides, the parents very much objected. We had, therefore, to do what we could by scraping and erasing the diseased parts. Things are, however, not looking very favourably; no doubt there is tubercular granulation tissue existing there, and I am afraid we shall have to resort to radical means before long.

Here is a little boy who came in with superficial caries of the outer metatarsal. That was scraped, cleaned, and rubbed with Chloride of Zinc; but here you will see the usual further development of this affection, spreading to the bones of the tarsus. There is a puffy swelling which I have no doubt means a considerable amount of tarsal bone im-

plication which will have to be dealt with more thoroughly, the case being past the stage at which rest will do good. You see here the prominent blue veins which almost always occur in cases of tarsal joint disease with the feeble atrophied condition of the parts beyond. It is not so marked here as in cases that begin in the astragalus. Here the swelling extends down to the toes, but where the disease commences in the astragalus and the puffiness is confined to the upper part of the foot, the atrophied condition of the limb is more marked.

Here is a boy of 7 who has been in here for a long time with hip-joint disease. It has not done badly, but we have been thinking that probably there is now an abscess. That is more evident to-day than when I last saw the case. These abscesses when once they begin to develop, do so very rapidly. There is evidently an abscess here over the groin, indicated by a general roundness, some heat, and, I think, some tenderness, though only a few days ago there was neither tenderness nor fluctuation. Even now we cannot make out fluctuation distinctly. My rule is to cut down directly I can make sure there is fluctuation. Although I may be pretty confident it exists, unless I can actually make it out with my fingers I do not incise. It is very difficult to detect early suppuration in the upper part of the thigh; because in these cases, where the muscles are left at rest and there is no resisting power, they fluctuate almost identically with an abscess.

In this other case, for instance, it was almost impossible to detect it. It is a particularly interesting case to me, inasmuch it is rather against my own theory. He is a lad of asthmous type as could be who came in here with hip disease. He had all the symptoms of it for a long time. The temperature chart showed that there was matter forming somewhere or other, but we could not detect it even with the most careful examination under an anæsthetic. So certain was I that matter must be forming, guided, of course, by the temperature chart, that I cut down upon the joint by the anterior incision. In this I acted rather against my own experience; it is only my own, perhaps; but still I have not been fortunate in cases in which I have excised the hip-joint from the front. Some have done well, but more have done badly, although I have followed all the injunctions of those who advocate this form of operation. But here I was rather

exploring for matter than intending to excise. Although, however, I found hardly any matter at all, I found a little caseous pus in the hip-joint, and a very necrosed condition of the head of the bone. That I removed with the gouge, and, as far as one's finger could tell, I left a perfectly healthy surface of the neck of the femur behind. Since that, I am pleased to say, the whole thing has gone on very well. We left a drainage tube in for a short time; through that a little matter came away; a little matter has occasionally come out from below since that was removed, but there have been no indications whatever of anything of the character of an accumulation. The boy has gained much in general health, and now there is, we may say, nothing but a superficial wound, and, of course, the process of healing that is taking place between the two surfaces.

Here is a boy with caries in his lower dorsal and upper lumbar vertebræ. The prominence, as usual in the lumbar region, is very slight. Several of us examined him without an anæsthetic and thought we could not make out any definite collection of pus. To clear away all doubt we anæsthetized him, and pus was then found on the right side deeply seated just below Poupart's ligament. Cutting down very carefully, because it was extremely limited, one came down upon a small collection of pus which passed underneath from the original seat of the disease. Just as preparations were being made to remove the boy, it was suggested there might be some on the other side. Under the anæsthetic we were able to detect very indefinitely some fluctuation very deep down in the pelvis on the left side. Cutting down upon that we opened a very early condition of abscess and got some pus out, which, of course, had not extended so far as on the other side. The case has done exceedingly well. Both these abscesses have closed. This is a good example of the benefits of a very early opening. That on the left side would, of course, have followed the same course as the abscess on the other, causing a long period of suffering. All this has been obviated by evacuating early, and both abscesses have now healed.

This other case of spinal caries is an interesting one, because of the course the matter took. The boy was admitted as a case of hip-joint disease, and many circumstances pointed to that diagnosis. But when he was undressed there was obvious caries of the lumbar vertebræ. The movement of

the hip-joint was, to a certain extent, limited, pointing to the probability of disease there. On going into the case carefully, however, we found that under an anæsthetic the movements of the joint were perfectly free, and that the trouble was due to the fact that the caries in the lumbar region had given rise to pus which, passing down through the sacro-sciatic notch, had presented on the back of the hip and travelled round the joint so as to cause inflammation of the muscles and fascia in its neighbourhood, thus giving rise to all the appearances of hip-joint disease. Of course, we are not sure now that the hip-joint may not become implicated by extension; but certainly that was his condition when he first came in. He has improved so far that the discharge is very much less, and we hope to send him out before long.

ON THE FEEDING OF PATIENTS AFTER ABDOMINAL SECTION.*

By ALBAN DORAN, F.R.C.S.,

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CARE in after-treatment accounts to a large extent for the so-called "triumphs" of modern surgery. In the early part of this century there were British surgeons who were famed for the dexterity with which they performed major operations. Yet their mortality was very high, as is testified in old text-books, for not only were anti-septic precautions unknown, but the after-treatment was very faulty.

Abdominal section, especially, requires careful after-treatment. Diet is always important after any operation, but especially when the alimentary canal and peritoneum have been disturbed. The removal of large tumours and the separation of adhesions involve damage to the peritoneum and disturbance of intestine, so that both require rest for repairs—a rest which can never be absolute. Abdominal operations usually cause a considerable amount of shock, during which it is important to maintain the patient's strength. Hence, when abdominal sections have been performed, the surgeon must ascertain how shock may best be counter-

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acted by dietetic, as well as other, means; how much the stomach and intestine can bear, and how far the peritoneum and intestines can be left undisturbed. My observations particularly refer to the removal of large abdominal tumours, and diseased appendages in women.

When a healthy person has been subjected to a simple, uncomplicated ovariectomy or hysterectomy, experience shows that the amount of shock is but trifling. There is no necessity for the immediate administration of stimulants or nutrient enemata. The case is altered when the patient is feeble or very old, or when the operation has been prolonged and complicated. The incapacity of the stomach to tolerate food soon after operation and the dangers of flatulence lead us to consider nutrient enemata. Whether they be used or not, the patient must take food sooner or later. What then is the best food to begin with? Is milk excellent or objectionable? Are meat-broths better? What fluids should be taken? When may solid nourishment be commenced? The peritoneum needs rest, so do the bowels. In this respect flatulence and the accumulation of scybala must be avoided. These conditions must be reviewed in detail. General care and prudence in dealing with individual cases will ensure the best results, hence we see patients recover after treatment on very different principles.

I have just observed how experience shows that in young and healthy subjects the amount of shock after some of the simpler abdominal operations is trifling. Such patients bear a twelve hours' fast very well. Indeed, feeding by the mouth often sets up and always increases vomiting. Beginners, being naturally anxious, are often deceived by appearances, so as to think that the patient is worse than is really the case. Pallor is common, but does not in itself imply that brandy must be given. Nervous impressions, as well as hæmorrhage and sepsis, greatly influence the pulse, urine, and cutaneous secretion. With ordinary care, the surgeon can distinguish trifling from serious symptoms, and can tell when he may safely leave his patient alone for twelve hours. Above all, let him avoid Opium and brandy.

A patient, however, may be in real danger from shock. Women over 60, or over 50 if poor, feel shock easily. Anæmic girls suffer in the same manner. Operations involving the separation of extensive adhesions, the removal of large, solid tumours, the disturbance of abscesses and septic

foci, or damage to important viscera are always followed by more or less severe shock. The patient is returned to bed weak and chilly; artificial warmth counteracts chill, but the surgeon must never lose sight of the fact that the debility caused by the shock is aggravated by fasting. A robust young woman is perhaps the better for a twelve hours' fast; to a weak subject, such prolonged abstinence is absolutely perilous. In such cases nutrient enemata are called for.* Only last January I removed an ovarian tumour from a woman, aged 67. On awaking from the anæsthetic, very slight restlessness was observed; a beef tea enema was given, and repeated every three hours at first, and every four hours later, till the fourth day. The patient spent the greater part of that time asleep. I have repeatedly seen similar good results in aged subjects. The operator should not give alcohol in place of nourishment. It only stimulates for a short time, leaving the patient weaker and less fit to assimilate real food. A healthy man who drinks at night not rarely fails to retain his breakfast next morning. A patient's stomach after operation is still weaker. I have known obstinate vomiting to follow the free administration of alcohol after operation, and to last for several days. The cases where Opium and stimulants are needed are exceptional, but this is a therapeutic question.

Rectal feeding is not only valuable in counteracting shock. It is also of service in cases where the patient suffers severely from nausea, vomiting, or flatulence, especially gastric flatulence with hiccough. These complications increase the patient's debility, and make the stomach irritable and slow to tolerate food. Enemata feed the patient, allow the stomach to rest, and thus make it all the sooner ready to resume its functions. An empty stomach is irritable after shock and fasting, but the irritability ceases when the subject is fed by enemata, though it would be increased by direct feeding.

On the direct treatment of the irritable stomach I need not dwell; I question whether draughts of warm water are advisable except to act as an

* Three ounces of beef tea, prepared without salt, are to be added to an ounce of hot water. The enema must be comfortably warm, about 100°; if too cold or too hot, it will not be retained. When there is no good cook at hand, the beef tea may be made by dissolving a drachm of Johnston's peptonized fluid beef in two ounces of boiling water, adding one ounce of cold water after solution. In cases of restlessness from pain, ten to twenty drops of Laudanum may be added to the enema.

emetic when the patient is troubled with retching. The followers of Mr. Tait's system are probably correct in discouraging the administration of liquids, on the theory that thirst allows the veins of the portal system a better chance of absorbing all peritoneal effusion. In complications evidently septic, food is not rarely tolerated by the stomach, which is then torpid rather than irritable. But foetid gases are very liable to collect in a torpid stomach, and to prove a direct source of danger to the patient. Hence rectal feeding is often advisable in cases where a septic focus is opened, where the intestines are distended with flatus, or where the temperature and pulse are high, to save the patient from overloading of the stomach. Here, as in all cases where the enema is used, great care must be taken lest foetid material collect in the rectum.

The nutrient enemata may always be discontinued when shock, sickness, and troublesome flatulence have passed off. I will not dwell at present on circumstances where the continuance of the enema is desirable. The next question is, what should be given when the patient is first fed by the mouth? It is best to make positive before negative statements, so I think it right to declare that I have ever found barley water* the best diet to begin with. I have watched over a thousand patients in the Samaritan Free Hospital, and noted that barley water is the diet most grateful to the majority. Milk when administered early after operation, often sets up flatulence, and if given as the chief staple of diet, it causes trouble when an action of the bowels is desired. Meat broths are nauseous to many patients. If taken by the mouth, they often disagree, and the urine tends to become very concentrated. This effect is much rarer when the same diet is given by the rectum. Altogether, broths and soups should not be taken until the bowels are opened. Barley water is nutritious and not unpleasant.

Operators are divided about the temperature of early liquid diet. I agree with those who object to the routine administration of cold or iced fluids. Every healthy tourist knows that taking excess of iced water or merely cold water makes the tongue dry and promotes flatulence. Some patients, however, cannot tolerate hot drinks, the same usually objecting, even more, to lukewarm fluids. Of

course, cold drinks should, or rather must, be given in these cases.

After forty-eight hours, milk may occasionally be added to the barley water. Only a few ounces should be allowed daily. I have already observed that milk often sets up flatulence and promotes the formation of scybala, but then I spoke of milk as the first diet given by the mouth. After the second or third day I have found that it answers well, provided that it be not made the exclusive diet; most assuredly, in my experience, it is better than broths or any other kind of animal food, before the bowels have acted. To counteract the tendency to the formation of scybala, oatmeal gruel* should be given at night, and small doses of Sulphate of Soda with Citrate of Potash, should be taken three or four times in twenty-four hours. I have seen scores of patients thriving well upon milk and barley water for a week, when these precautions were not neglected. When broth has been given and the urine is concentrated and full of uric acid, it becomes clear, bland, and of a lighter specific gravity when milk diet is enforced. Tea is not to be absolutely forbidden. Half a cup, weak, and taken with a little milk and no sugar, in the afternoon, after the third day, is very pleasant to the patient, and does no harm, provided that there are no signs of gastric irritation, nor any other bad complication. The operator must watch lest the tea cause harm, as not rarely happens. Much judgment is necessary in cases where patients have a deep prejudice against some particular food. No patient must be suffered to dictate, but forcing unpalatable diet upon her may set up much trouble. These prejudices are very frequent in cases of chronic disease of the appendages.

The date of opening the bowels is a question closely associated with the present subject, but I have not time to discuss it at length. I have always found that when the tongue is foul, and there are signs of other inconveniences due to simple constipation, a purgative acts best. The same applies to cases where the diet has clearly disagreed. In healthy patients, with a good appetite and a clean tongue, an Olive Oil injection, followed by a simple enema a few hours later, is preferable to purgatives, as the accumulation of

* Add two ounces of barley to a pint of water and simmer for an hour, not longer, else the mixture will become too thick for an invalid. Two ounces may be given every two hours.

* To make the gruel, boil a breakfast-cupful of milk, dissolve two tea-spoonfuls of fine oatmeal in cold milk and mix with the boiling milk; continue to boil for a few minutes longer. Coarse oatmeal will require longer boiling.

hard scybala is the only evil that has to be overcome in these favourable cases. The enema is also best in cases that are septic, or otherwise in real danger, as drugs are then apt to cause or increase flatulence. By drugs, I mean Liquorice Powder or Colocynth Pill. Castor Oil is open to objections. The administration of saline purgatives as the direct treatment of peritonitis is a therapeutical question of deep interest, but not intimately associated with the subject of diet.

After the bowels have acted satisfactorily, meat broths usually answer well as diet, and in a few days fish, later on fowl, and then meat may be taken. During the second week, however, farinaceous food, such as milk puddings, sago, and tapioca, should be preferred to animal diet. Provided that ordinary prudence be exercised, the diet question is not likely to give trouble after the first week.

We may conclude by considering how far the peritoneum and alimentary canal may be kept at rest whilst the patient is sufficiently fed, a problem already half solved in the above observations on diet. The nutrient enema involves the least possible disturbance and encourages the passage of flatus, especially if the nurse be careful about passing the rectal tube before the administration of the enema. Refuse must, at the same time, be washed away from the rectum, else it may do much harm, the least being painful irritability of the rectal mucous membrane. There is strong evidence that decomposing refuse may set up septic changes in structures near the bowel. The surgeon must never forget that flatulence after abdominal section is in itself a direct source of danger. Hence, all diet which causes flatulence is unsatisfactory. Gastric flatulence, if promptly treated, causes little or no trouble, but if food be persistently given by the mouth severe vomiting will follow, or the stomach will become greatly distended. This distension, like the equally well-known distension of the colon, sometimes sets up alarming symptoms which may cause the operator to suspect a deeper but imaginary complication, or may mask a real danger. Flatulent distension may be the result, or as some believe the cause, of peritonitis. In any case it aggravates that formidable disease. Flatulence may be the direct cause of fatal obstruction. It gravely complicates adhesions, of which it may be the result, but may also be the cause. Many competent observers hold that septicæmia itself is a direct result of obstruc-

tion. A segment of intestine is damaged by handling at the operation, and becomes paralyzed and distended with air, or becomes distended through the irritation of ingesta. This distension itself involves a certain amount of obstruction; then portions of food or fæcal matter and gas, retained in the affected segment of intestine, decompose and are absorbed by the blood-vessels, with serious results.

It is clear then that flatulence is the chief enemy to be taken into account when diet is prescribed. Small doses of soda water or Citrate of Potash sometimes counteract flatulence, but sometimes aggravate that symptom. Flatulence usually demands alteration in the diet itself. If persistent, it is advisable to return to nutrient enemata. I have taken this course, even after the first week, with the best results. When flatulence is present whilst nutrient enemata are being given, or is not diminished when diet is administered by the rectum, it is best to throw up hot water with Turpentine.

I have spoken chiefly of my own experience and that of my colleagues. There is little reason for me to criticize others, yet I must admit that I have known several different principles of diet much extolled by experienced operators, to answer very ill. Ice, champagne, and stimulants, are discarded by nearly all, if not all, British operators. The objections which I have heard raised against nutrient enemata seem to be frivolous. Of course, they must be properly made and carefully administered, nor must refuse be allowed to collect in the rectum. Exclusive milk diet or the total exclusion of milk as diet, seem to me equally objectionable. The forty-eight hours fast after operation involves much risk to weak or weakened patients. Those who wish to see how both sides of this question are treated by experienced writers should read Dr. Christopher Martin's work "The After-Treatment of Cases of Abdominal Section" and Dr. Byron Robinson's recent letter to the "Journal of the American Medical Association," February 10th, 1894, on "Fluid Drinks after Laparotomy."

I need hardly apologize for the amount of commonplace subjects which I have discussed. Full attention to after-treatment is one of the best ways to attain successful results; it is at first less interesting to contemplate than the question of antiseptics, short incision, and the drainage tube, but it is quite as important.

A LECTURE

ON

OZÆNA, OR ATROPHIC RHINITIS.

Delivered at King's College Hospital, Feb. 21, 1894,

By GREVILLE MACDONALD, M.D.,

Laryngologist to the Hospital, and Physician to the Hospital for Diseases of the Throat, Golden Square.

(Concluded from page 336.)

Another class of case presenting a great amount of dryness of the mucous membrane of the nose, is one which is the opposite of that I have just described. It is really a condition of inflammation in the mucous membrane of the nose, where the whole surface is heightened in colour, the erectile tissue by no means collapsed, but sometimes even engorged, but where the mucous membrane presents a dry appearance. The pharynx is also more or less dry, heightened in colour and glazed, and there may be considerable laryngitis. These cases are to be found, not in anæmic girls, but in middle-aged men who live not wisely but too well, and men whom one would be inclined to pronounce gouty, without any minute inquiry as to the ordinary indications of the accumulation of uric acid. They are over-fed men, and, for the most part, men who indulge too freely in alcoholic stimulants. I believe that the alcohol has more to do with it than anything else. In these patients you may find considerable increase in the amount of mucus in the nose, but dried up. You will find dried masses of mucus (not muco-pus) in films which stick for the most part on the septum, and also on the anterior part of the middle turbinated bodies. This accumulation of mucus on the septum excites a good deal of irritation in the nose; it sets up sneezing and tickling; and, unable to get properly rid of it by blowing, the patient resorts to the use of the finger to remove it. This is apt to produce a little abrasion of the dry mucous membrane, and induces epistaxis. This, by the way, is the commonest cause of nose-bleeding in any patient. After the production of this abrasion the mucus tends to accumulate there all the more, and adhere the more firmly. Next time it is removed more extensive abrasion is produced, and ultimately an ulcer may form. Next, the perichondrium may be exposed, after which ulceration of the cartilage may occur, then necrosis, and ultimately a perforation. These

cases, therefore, of simple dry rhinitis, without any muco-purulent discharge, without any ozæna, occurring in middle-aged men who over-drink and over-eat, are apt to be accompanied by epistaxis, and ulceration and perforation of the septum. It is frequently maintained that if a patient presents himself with perforation of the septum, you may diagnose the case as one of syphilitic origin without any further inquiry; but this is not so. By far the majority of cases of perforation of the triangular cartilage that occur are caused in the manner I have described, and are not the result of syphilitic disease.

In neither of these cases is there any interference with the sense of olfaction, nor any foetid odour; and you will see what a different picture they present to cases of typical atrophic rhinitis.

It is maintained by some most excellent authorities—chiefly German, and supported by many good men in America—that this atrophy is a later stage of hypertrophy. They tell us that hypertrophic rhinitis is a condition anterior to the atrophy I have been describing. But I think this opinion must be founded upon theoretical considerations. I do not think—and I have had considerable opportunities of observing these cases—that there is any clinical reason whatever for that assumption; and if you will just consider the classes of patients in whom we have hypertrophy and atrophy respectively, you will see there is no likelihood of one passing into the other. Hypertrophy of the erectile tissue of the nose is to be found for the most part in older patients, in men, and in patients who are at any rate well-nourished, and certainly not anæmic; while, on the other hand, the atrophic cases occur for the most part in anæmic people, in young girls, and people who present a distinctly strumous physiognomy. So that it is rather absurd to suppose that the hypertrophic condition will ever lead to the atrophic. I have had under observation some hypertrophic cases for many years, and have never seen the slightest tendency to atrophy in them; on the contrary, there is a tendency to increasing hypertrophy. The condition we do get anterior to the atrophy is a condition of chronic cold in the head, with some vascular engorgement of the erectile tissue. I believe it is that engorgement which these observers have considered to be identical with hypertrophy; but, for the reasons I gave you in a former lecture, you will see at once that this is a great clinical blunder to make.

The other conditions to be distinguished from atrophic rhinitis are other cases of suppuration. The presence of the large masses of inspissated muco-purulent secretion, the atrophy of the inferior turbinated bodies, generally speaking the symmetry of the disease, and your inability to find the pus flowing from any one particular region, will all make it impossible for you to mistake the one condition for the other. But sometimes when there is some suppurating mischief high up in the nose, high up, for example, in the middle or superior meatus, we have not only a flow of pus from the upper regions, but a good deal of mucus along with it, which may tend to dry up as it reaches the inferior meatus, and accumulate there, so giving the appearance of atrophic rhinitis. When the discharge is all pus it has no tendency to dry up in crusts; only when mucus is mixed with it does that occur: but in such a case we shall be led to a correct diagnosis by observing that, generally speaking, the mischief will be unilateral, that there will be a good deal of unmistakable pus as well as the crusts of muco-pus, and that there is little or no atrophy of the inferior turbinated body.

If we are in any doubt, we should examine the region from which the pus is coming. If with a probe, we find any caries, or the presence of a sequestrum, rhinolith, or other foreign body, our diagnosis will be made. Generally speaking, when the discharge seems to be flowing from a definite quarter, we may dismiss the probability of atrophic rhinitis. Thus you will see there is no real difficulty in making a diagnosis.

The prognosis will depend entirely upon whether we are competent to treat the disease successfully or not. I think you will find it commonly held that the disease is incurable. Most of the patients certainly come to one with the belief that nothing can do them any good; and, generally speaking, most of them have had a good deal of treatment at other hands, in the course of which many remedies have been tried and not to much purpose; so that it is not to be wondered at that the patients labour under this hopeless view of their case. But the fact that the disease has a tendency to spontaneous improvement as the patient grows older, together with the fact that the patient always feels better as long as the nose is properly washed, might lead to the belief that more thorough, systematic, and scientific treatment would produce a tendency to more rapid improvement. Provided we do the best possible

for our patients, I think we shall be able to justify my claim that the disease really is curable.

Of course the structural atrophy cannot be remedied; but we can overcome the worst symptom of the disease, that is to say, we can make the secretion healthy and prevent its stagnation and putrefaction. What then are we to do for it? The only treatment of any avail is to treat it on common-sense principles. We must keep the nose clean; and therein lies the whole difficulty of the matter. I do not think it of much importance whether we use this or that remedy, whether we administer this or that tonic, whether we keep the patient at the seaside or allow her to remain in an unhealthy, damp locality; it all matters comparatively little, provided we can make and keep the nose clean. To ensure this we have to see the patient ourselves very frequently. In the first place it is necessary that we should be perfectly familiar with the normal appearance of the nose, and be able to examine it thoroughly well with a reflected light and a speculum. I want to emphasize the difficulties in treating these cases. Even though one be as energetic as possible, failure may ensue from inability to illuminate the interior of the nose properly, and to manipulate instruments in the interior of the nose. The first requisite, then, is a thorough acquaintance with the use of the speculum, and with the use of a probe in the interior of the nose, so that we may be able to apply our instruments, whatever we use, to this, that, or the other region with the minimum amount of suffering to the patient, and the maximum effect. The best way to clear the nose of the accumulations is by a stream of water. The patient, covered up with towels, is set in front of the operator, who, if he has any respect for his own clothes, also covers himself with a towel. He has a large basin of warm water to which may be added a little Permanganate of Potash, Peroxide of Hydrogen, or some other oxidising agent, to lessen the fœtor. The patient is given a basin; and with a speculum and a light thrown right into the nose to guide him, the operator takes a syringe with a fine nozzle full of the warm water, and directs a stream in this or that direction wherever the crusts accumulate. The washing must be proceeded with until every particle of secretion is removed.

The ease with which this can be done is surprising, provided we have a little skill. The very roominess of the nasal fossæ facilitates the pro-

cess. At first you see the crusts sticking about everywhere, and you may be puzzled to know what some of the regions are, so different from the normal appearance do they seem. As soon as every particle of accumulation is removed we find that the fœtor has vanished. The next thing to do is to obviate further accumulation, and the best way to secure this is to prevent the patient breathing through the nose, so that the inspired air may not dry up the accumulating mucus. For this purpose the nose is plugged with a good big piece of cotton wool, which the patient is directed to retain as long as possible. After a time it feels so irksome that she says she cannot breathe and pulls it out. This we must expect, but should endeavour to persuade her against it. The cotton wool may be impregnated with some stimulating substance so that the mucous membrane with which it is in contact may be stimulated to a healthier action. Cotton wool saturated with Boracic Acid is good, or with Eucalyptus is particularly good; but the medicament I like best is the Ammonio-chloride of Mercury, 2 per cent. in wool. It acts as an admirable stimulant, and is antiseptic; and if the patient should get a little into the pharynx and swallow it, it will do no harm. To obtain the full action of the drug we should pass a big plug along the inferior meatus. The plug should be two or three inches long, and as thick as can be forced in with a probe. After a while the patient can be taught to insert the plug herself. The patient must be seen frequently, if possible every day to begin with, so as to wash away any fresh accumulations that may occur. After daily treatment for a week or two we may see the patient at increasing intervals, allowing her meanwhile to carry out the washing herself. One of the first cases of the sort I have ever had, I have had under observation on and off for some eight years. The patient considers herself perfectly cured; she has no symptoms now, she has perfectly recovered her sense of smell, there is never any muco-purulent discharge, although there is occasionally a little drying up of the mucus from the wideness of the nasal fossæ; but I cannot, of course, consider her cured, as irremediable mischief has been wrought by the disease. But the atrophy is of little importance so long as there are no symptoms.

That, then, is the principle of treatment I recommend. I am prepared to maintain that the condition is practically curable if only we treat it correctly. I am tired of hearing of new remedies

called cures for the disease. I think you may take it as a general principle in therapeutics that the greater the number of remedies supposed to cure a disease the less is the curability of that disease. If we limit ourselves to water and cotton wool in these cases we shall produce a cure without resorting to any of the thousand and one more or less useless remedies advocated as infallible cures.

THERAPEUTICAL NOTE.

Chloride of Ammonia in Renal Disease.—Corrie finds Chloride of Ammonia an excellent remedy in cystitis. He prescribes ordinarily a No. 1 capsule of Squibbs's pulverized purified Ammonium Chloride, to be taken three or four times in the twenty-four hours, preferably when the stomach is somewhat empty, each dose to be followed immediately by half a goblet or a goblet of pure cold water. The following are some of the conditions in which the drug has been given faithful trial, with most satisfactory results in *every instance*:—

Cystitis dependent upon stone in the bladder, stricture, hypertrophy of the prostate; deposits of urates, etc.; gonorrhœa (male and female); cystic irritation from uterine disease or menstrual disorders, malarial disease, masturbation, early pregnancy, simple urethritis (traumatic) in newly-married women; cystic and renal sequelæ of *la grippe*.

In the majority of cases it was simply surprising to note the rapidity with which the urine was cleared of bladder-mucus, blood-corpuscles, pus-corpuscles, urates, phosphates, etc., the distressing symptoms disappearing therewith; and in no case did the salt occasion any gastric or other disturbance when taken as ordered. No explanation of the *modus operandi* of the remedy is offered. The capsules are to be filled only as needed for administration, as the salt dissolves the gelatin in a short time.—(*Virginia Med. Monthly*).

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A LECTURE

ON

ANGINA PECTORIS.*

By JAMES F. GOODHART, M.D., F.R.C.P.,

Physician to Guy's Hospital.

THERE came to me, the other day, a gentleman of sixty years of age, with the following history: He had lived a very active life both mentally and physically. He had carried on a large private school, and, although he did not know that it had told upon him, he had, through many years, been exposed to the usual worries incidental to such a position, and they, of course, had not been few. In August, 1891, after playing golf, his back became stiff, and this seemed to oppress him generally. In November of the same year he was out shooting with a medical friend, who, noticing that his breath was short, sent him to a physician in town who said he was suffering from pseudo angina; gave him Nitro-glycerine, which relieved him; and he then remained well for some time. But he had a return of his symptoms, and on and off this continued to be the case, and he and his wife thought this was invariably so when he had been worried or overworked, or when his digestion or liver had been upset, which latter meant, when his bowels were confined. He saw another well-known physician in the spring of this year, and he also (I suspect from motives of humanity) called it a false angina, treated him, on general principles, by laxatives and tonics, but, in this instance, without any definite result. Anyhow, for the last six months he has become decidedly worse, and finding that anything excited or worried him with increasing readiness, he has given up his work, and taken his passage for Australia to see his relatives out there. But before starting, by the advice of an old medical friend of his, he came to see me. He was clearly excessively nervous, grey, but not unhealthy looking. He had a fresh colour, but was not plethoric. I sat and talked with him for

half an hour or more, and during that time he had several of his attacks. In them he put his hand to his sternum to press it, saying, "Oh! here it comes, now it is very bad," and then, in a few seconds, "There! now I am all right again." His face flushed decidedly in the paroxysm, but he did not sweat, his pulse never altered a bit—and I sat with my hand on it nearly the whole of his visit on purpose to make the observation. It is certainly not a hard pulse. His urine, of which two specimens were examined, was in each case high coloured, and of sp. gr. 1030. It contained neither albumen nor sugar, nor any uric acid after standing. The heart was to all means of examination perfectly healthy, and upon a second examination I made this note, that "I am perfectly certain that if he went to an insurance office, and said nothing, he would be passed as perfectly sound."

One other point, I may mention that his attacks are always worse when his bowels are inactive; and yet another, that they are always more frequent after his dinner in the evening.

I told his wife that I had no doubt whatever that the disease was true angina, but that angina was not always so deadly as it has been pictured and described, and that in *any* case where the history spread over so long a period as this, and when the attacks frequently recurred, yet were of a mild character, the outlook was distinctly favourable.

It is this forecast that I would enlarge upon and justify. In this particular case I acted up to my opinion. Coming, as he did in a sense, to the arbiter of his destiny, and prepared as he was to make a considerable sacrifice if it should be determined that it was not safe for him to go to Australia, I said "you had better go," and I trust by this time he has safely reached his destination. Ten days had to elapse before he sailed, and I told him to go back at once to the Nitro-glycerine tabloids that had done him good before, and in consideration of the high sp. gr. of his urine, I also gave him some Iodide of Potassium to take three times a day, and I heard, before starting, that he had been greatly better.

Let this case, then, be my text. But in dealing with the subject of angina pectoris I must first say something upon the matter of its pathology. You

* The substance of a Clinical Lecture delivered at Guy's Hospital, and of a Paper read before the South-West London Medical Society.

will remember that of late years this has been attributed, I think I may almost say exclusively so, to a sudden rise of tension in the arterial system. Dr. Lauder Brunton demonstrated, now many years ago, in a case of aortic disease with angina, that the attacks were associated with if not preceded by a rise of the arterial tension; and that by paralyzing the arterial spasm by Nitrite of Amyl the pain was relieved. But the now well tested assertion that Nitrite of Amyl and Nitro-glycerine are, of all remedies, the most generally serviceable in this disease, has led many to accept the condition upon which their use was founded as true for all cases. The late Dr. Hilton Fagge, while endorsing this argument, adds, however, with characteristic caution, that he is not aware that this increase of tension has been found to exist in all cases.

Now, that high tension in the systemic arteries is associated with angina in many cases is true; and is, moreover, an important fact to remember in the treatment of all such cases. The following illustration will go to show this:

A young man of 32, an exceptionally early age I may remark, to have angina except in association with severe disease of the heart, was admitted into Guy's Hospital suffering from what appeared to be an attack of severe angina. He appeared to be very ill, and was of an ashy grey colour, with intense pain in the epigastrium. He had been suddenly attacked a week before by a severe pang in the chest, as of a knife running him through, and he was unable to get his breath. It lasted for some hours and then subsided. He remained in bed till the next day, but on then getting up his pain speedily came on, and obliged him to return to bed, and for the next three days he had returns of less severity, till at last it went. A second attack came on just before he applied for admission, when he appeared in the most intense distress, taking no notice of his surroundings, unable to lie down, sitting forwards, hands on knees, gasping for breath, but with a pulse of only 60, and no difficulty of respiration. Not having seen the attack, and he being so young, I was inclined to make light of the angina, but on going over him it was found that his accessible arteries were unusually thick, and his pulse very hard, and so he remained; his urine being of low sp. gr. I therefore came to the conclusion that the case was one of those uncommon ones where arterio-capillary changes had taken place early in life, and that he was suffering from the angina of high tension. He was much

relieved by Nitro-glycerine. I may follow up this case with the remark that the history of chronic Bright's disease would also supply many cases of angina, and point to the same conclusion. So, also, does the occurrence of angina in any state of enlargement of the left ventricle. And I think, too, that I have seen true angina produced in a case of extreme dilatation of the right ventricle, when the symptoms were those of pure pulmonary obstruction also. All these seem to me to be appropriately explained on the hypothesis that the pain is produced by, or, at any rate, is the outcome of, muscular fatigue. Tire out the heart muscle, under certain circumstances, or perhaps in particular individuals, and angina results; just as under certain circumstances, or in particular individuals, cramp in the calf will attack the muscles of that region as the result of fatigue there. And it has been proposed, indeed, with much plausibility, to consider angina pectoris as a heart cramp, of similar kind to that other cramp which many of us no doubt know so well.

But that high tension is the sole cause, or even the usual cause, of some of the most typical cases I am quite certain is not the case. For, in the first place, excessive tension is a vascular condition of exceeding commonness, and although angina pectoris cannot, unhappily, be said to be *rare*, yet its prevalence will, by no means, compare with the exceeding frequency of sudden rises of tension. It ought, indeed, did it own such a cause, to be tenfold more common than it is. In the next place, I have now had several opportunities of having my finger upon the pulse during a spasm of bad angina, and more than once I have had a finger on the radial in a case of angina pectoris from the beginning to the end of several attacks of pain. And I am certain that there has been no excess of tension of any kind; on the contrary, the pulse has been feeble throughout, and has not altered or faltered in any way. I believe that there are many cases of angina in which you can get no indication of this kind, or indeed, of any other, from the pulse, of any value, of the gravity of the attack through which the patient is passing. It is difficult to think that such could be the case if the rise of tension were the cause of the disease.

Then, again, the actual conditions found upon the post mortem table are worth consideration. In quite a respectable number of cases the coronary arteries have been found diseased and

plugged with old clot; and the heart muscle has in several instances been found so soft and fatty that it was lacerated with the greatest ease,—a condition that is absolutely opposed to the existence of a tension that requires as one of its conditions of existence the integrity of the cardiac muscle.

A somewhat similar difficulty applies to the idea that the disease may be a severe form of cramp of the muscular wall of the heart. One could surmount that created by the fact that in many cases of very serious disease of the heart-muscle there is absolutely no pain, because muscle pain is peculiar in the special conditions that call it forth. I suppose, that in order to fulfil its function, it would hardly do to endow muscle with *common* sensation, and therefore it has its own special muscular sense. But overstep the limits of its tolerance, it might be said, and this insensibility no longer applies, the worm turns with a vengeance, and we then have that most agonizing of all pains, a muscular cramp. But anyone who has felt the quiver and the throb of the calf in a state of cramp will find it equally difficult to believe that such a condition could affect the *heart*, and yet leave no trace upon the *pulse*. One could readily understand, however, that if it did, it would put an end to existence with tragic swiftness. And to these somewhat exceptional cases of very sudden death in angina such an hypothesis might still be considered to apply.

It will be apparent, therefore, that some other explanation is wanting for the residue, a large residue, of cases. And I can offer you none that fills the gap better than the one originally suggested by Trousseau that the disease is a neuralgia. Epileptiform neuralgia, he termed it, meaning by that a disease that owned no definite morbid anatomy, and that showed an inveterate tendency to repeat itself. It may be said by those who want a definite lesion for every disease that a neuralgia will explain anything and everything. But be that as it may, the group of affections that cannot be called anything else, is a large one, and a very real one in our list of diseases at the present day.

And this neuralgic hypothesis has, the more one thinks about it, the more to be said in its favour. In the first place, many, *very many*, so suffering are conspicuously nervous. The case given at the outset is an instance of this; some of them are so much so that they have resembled more the

condition of Graves's disease than anything else in their tremulous excitement.

Next, it comes and it goes. Goes, perhaps, for years; perhaps for ever. And although I am far from saying, or thinking, that a muscular change may not exist and yet repair itself, for I believe that this must be the case in some instances, still such an explanation hardly applies to all cases, the coming and the going are so sudden, and the latter may be so complete.

Again, I have to say that there are processes of the nature of neuralgia which are analogous to this, let us call it, thoracic neuralgia. I refer to the gastric crises, and the lightning pains of *tabes dorsalis*; the destructive pains that usher in diabetic coma; and with this, I think, may be coupled that condition which has been called gout in the stomach, and which I take to be an abdominal crisis of like kind, although I am aware that in one or two recorded cases the actual condition found at death has been some serious lesion of the stomach.

Then, too, I think that the common consensus of opinion that angina pectoris and gout are closely associated—an opinion that I most decidedly share—is another point in favour of the disease being neuralgic in character. I believe there is a very close association of the one disease, if not with any classical form of the other, at any rate with conditions that may be said to *breed* gout; and to be presumptive evidence in its favour. I am entirely of opinion from what I have seen, that angina pectoris is a risk attaching to full or free living, particularly in the sedentary by habit or occupation.

Lastly, I may point to a result of the paroxysm, which is in no small number of the severe cases, not only a severe pang, but also a rapid production of what, to all intents and purposes, is an acute bronchitis, often with the expectoration of blood in the form of a rusty expectoration. A symptom this, by the by, that I am accustomed to teach, is valuable for prognosis, as it is generally one of the worst possible significance. Now, this bronchitis or cedema, or whatever it be, might seem more readily explained by a stasis of the circulation due to heart-failure, than by any other cause. And so, perhaps, it is. But against that view it may be urged, as I have done with regard to any exclusive holding of the theory of heart cramp, that it is difficult to conceive of the production of any serious pulmonary stasis

such as this *so rapidly*, while all the time the heart's action and the pulse may have altered not at all.

Now, I have spun out this somewhat lengthy argument, in this somewhat dreary manner, because, in a malady that is so obscure it is really of importance, both as regards prognosis and treatment.

It is easy enough to say "Here is a case of angina pectoris—we must give Amyl Nitrite,"—or Nitro-glycerine, as the case may be, and I do not know that after all I can add much to this practical commonplace. But yet I think, from a study of the disease such as this, one does really *seem* to have a little more insight into this terrible affection, and if so we shall probably have a little more power in our method and manner of dealing with it.

With special reference, then, to prognosis and treatment I would sum up the causes of angina pectoris thus:—

First, and most rarely, possibly only in the comparatively few cases that die straight off—and not in all of those, for actual lesions—tears and extravasations, for instance—in degenerate muscle, must take their share in the production of suddenly fatal angina—there may be a *heart cramp*, such as one meets with in the extremities,—the leg and foot, of course, more particularly. For these the prognosis is well-nigh hopeless and the remedies clearly Nitrite of Amyl and Nitro-glycerine given freely, to arrest, not the arterial tension, which does not exist, according to my thinking, but the muscular spasm. Morphia will also still find a fitting subordinate place in the relief of pain, but its use is too often crippled by the severity of the symptom, and it is notorious that it has often been given in enormous doses without benefit.

I will place next, because next in fatality, the angina of old people, and which is, whatever the other cases may be, essentially the angina of degenerate muscle, fatty or fibroid or granular as the case may be, and often with thrombosis of the coronary arteries. These cannot be very encouraging; but even in these conditions, if they should occur, any of them, in younger people, as disease of the coronary vessels sometimes does, one may hope on in the light of the teaching of recent years, viz., that the coronary vessels anastomose freely. It was always taught otherwise, but Wickham Legge and others have de-

monstrated that this is wrong; and I have myself repeated the experiments, and it is easy enough to fill the branches of the one coronary vessel from the other. I am, therefore, in these cases especially careful to insist upon the most absolute rest, in the hope that, with time given, the diseased vessel or vessels may re-establish its current by collateral channels, and by so doing give a chance to the diseased muscle to rehabilitate itself.

Thirdly, there is the group, and no doubt a large one, where the vascular tension is in excess, and the muscular wall of the heart fails in consequence. In these the prognosis is hopeful in proportion as the cause of the tension can either be reduced or removed. Nitro-glycerine is again very useful, perhaps more so than Nitrite of Amyl, because its effects are more enduring; (and because less now applicable to any state of cramp; more, to its action upon the systemic arterioles). But it is worth while to say that while using these comparatively new remedies we must not forget our old fashioned friends, viz., Blue Pill, smart purges, saline diuretics; and, watching their action on the case, the various heart tonics.

Lastly, there comes the neuralgic group, which to my mind possesses the greatest interest of all, first: because, in the purely neurotic, if the attacks come and go, and are of mild degree, one may, I think, take a fairly hopeful outlook. Next, because they are much helped by encouragement, by *facing* the question of angina, *if they have it in mind*, and explaining things to them. In these cases, too, the attack is so often independent of *exertion*, more dependent upon excitement or worry. Therefore, moderate exercise can be allowed them, and does good. Next, because such cases go on for years, perhaps recovering completely so far as we can see, perhaps now and then from various causes having a recurrence of mild attacks, which pass away under treatment. Next, because so many of these cases seem to me to suggest gout as an element in the cause, and they are sometimes much relieved by the administration of Iodide of Potassium. I remember seeing a case of this sort in a youngish man of about 45; active in mind, but taking little exercise. He had two or three attacks of what seemed to be severe angina, and he appeared to be completely and quickly relieved, by this drug; and he has now remained well, I believe, for a long time. In these cases, too, the judicious use of Blue Pill and saline aperients will probably help a good

deal by oiling the wheels of excreting organs. But when one introduces the question of the treatment of the gouty diathesis, I open, indeed, the floodgates of discussion. Even the youngest of us can plume his wings of speech by such a lubricant as gout.

But I will make just this further remark about the treatment of such cases as the gouty, viz., that for each case we should endeavour to find out what is the diet appropriate to or that best suits the case. We all know that one has only to call a case one of gouty something, to suggest the prescription of a diet that is certainly robbed of all its carnal charm,—to that I do not so much object, for I remember to have learned with much pain, now many years ago, from Henry's "First Latin Book," that *Homo edit ut vivat non vivit ut edat*,—(I am afraid that this is hardly a Ciceronian aphorism)—but it is also robbed of much of its nutritive value. Now nutritive value is known by certain gross and vulgar characters, such as physiological chemistry has made us all well aware of. But nutritive value has also its *select* and *individual* side, than which nothing can be more perplexing, and often apparently contradictory. And in no people do you see these differences more pronounced than in the group that is ticketed in common gouty. Over and over again you have seen a gouty man being starved because he had uric acid in his urine, and was supposed therefore to be gouty. We all of us admit the impeachment as against ourselves, because we were taught to do so by those who were wiser and more experienced than we. But what is uric acid but the ash of many a burnt-out fire? These cases seldom get their individuality studied; but obviously it makes all the difference in the advice to be given, whether the man be thin or stout, whether he be a free liver, or a spare feeder or drinker, whether he be a hard brain worker or whether he is one of those happy individuals who know not the pain of brain fag; whether he be a man easily affected by worry or can let care settle with the jauntiness of unconcern. Now I venture to say that angina in the gouty requires our most penetrating attention to these matters, and there are those who require, even, it may be, in excess of their usual habit, perhaps one of those very things that in the unwisdom of routine we might be inclined to cut them off. Certainly there are some to whom port wine, champagne, or cream, or butter, or sweet-stuff,—to name the reputedly most obnoxious

articles that occur to me at the moment, will, each, in its place, and opportunity, bring healing on its wings. But, unfortunately, life is not long enough to allow any individual to formulate rules that are ample enough for the many, and, at critical moments in our career, individuality has to be read by the key to someone else's cypher, and the result, needless to say, is not always satisfactory.

And this leads on to another point, viz., how difficult is angina often to be *sure* of, and it is so by reason of its exceeding commonness, and also by the numerous counterfeit presentments it appears in. Thus it comes in the garb of all sorts of symptoms; sometimes as flatulence; sometimes as stitch in the side; sometimes as colic; and so on through a very long list. But in these cases the disease is mostly of a mild order. Yet not always. I have notes of cases that illustrate this all too sadly. Here is one.

A lady of 46, was sent to me with this history: She had been under treatment for a fortnight or so with a most puzzling and intractable pain which had posed as a gastralgia—it came as a sort of burning at the epigastrium and round under the *right* ribs, and then down the right arm—a description that might well have indicated an hepatic colic. She was a very nervous, and yet as is often the case, a very sensible woman. She told me that the pain seized her suddenly in the chest, and she fell flat on the floor, evidently, therefore, it was very severe. It lasted ten minutes, and she broke into a profuse perspiration. It came quite suddenly, and as far as she knows causelessly, and has repeated itself at all sorts of times, waking her out of sleep even. But she did not think exertion affected it. She looked a wonderfully healthy woman. As I could find no evidence of disease, I concluded it must be angina, and a few days after she died suddenly in an attack. A post mortem examination was made, and no disease discovered.

In conclusion, I must now say that after all one's endeavours to differentiate between various *causes*, and between severe cases and mild ones, it must still be confessed that angina pectoris is a disease that will sometimes sadly belittle our would-be wisdom, by overturning a favourable forecast with crushing suddenness and often enough too by defying the most careful and attentive treatment.

A CLINICAL LECTURE ON SOME POINTS CONNECTED WITH CONCUSSION OF THE BRAIN.

Delivered at St. George's Hospital, Jan. 30, 1894,

By **WILLIAM H. BENNETT, F.R.C.S.,**

Surgeon to the Hospital.

GENTLEMEN,—Cases of concussion of the brain are as you know common enough both in hospital and private practice, so common, in fact, that many of you, when such cases present themselves in the wards, are apt to look upon them as very uninteresting, and fail to pay them the amount of attention which they merit.

I assume, for the time being, that you are conversant with the typical symptoms of concussion of the brain as described in the text-books. It is not my intention, therefore, to dwell upon them in detail, for I always endeavour in these lectures to draw your attention to points which, although they are learnt by experience, do not always receive sufficient emphasis in the works which as students you commonly use.

For the purpose I have in view to-day the cases of concussion, as we commonly see them, may be divided into four classes:—

1. Those in which the loss of consciousness lasts for an hour and upwards.
2. Those in which the loss of consciousness lasts for, say, from ten minutes to an hour.
(These two classes go fairly well together, because they are both of a severe type.)
3. Those in which the loss of consciousness extends from a few seconds to a few minutes; and
4. Those in which there is no real loss of consciousness at all; this last class being, for my present object, the most important of all.

It is at first sight a singular thing—and you will notice this especially in private practice, because there is then a better opportunity for keeping in touch with patients after they have recovered or are supposed to have recovered—that the cases you hear most of subsequently are as a rule not those which originally were most severe, but the comparatively trivial cases such as those I have

mentioned in classes 3 and 4, the cases, that is, in which the loss of consciousness has lasted for only a few seconds or two or three minutes, or perhaps in which there has really been no loss of consciousness at all.

Here I must digress for a moment to explain exactly what I mean by a case of concussion of the brain in which there is no loss of consciousness; because of course you know from your text-books that the crucial symptom insisted upon in cases of concussion of the brain is the insensibility. But this insensibility is, after all, only a matter of degree; and there is a condition produced by a concussion of the brain which does not amount to anything like real unconsciousness. The patient, in point of fact, does not necessarily lose his senses entirely, and may be quite able to recollect the incidents as they occurred at the time of the accident. Such cases are very common indeed in the hunting field. A man, for instance, riding under the branch of a tree may get a blow on the head; for a second or two he is rendered giddy, reels a little, perhaps, and almost falls off his horse, but recovers himself, and goes on possibly for the rest of the day. Thus the effect produced under such circumstances may be merely a kind of general giddiness which for the moment, without exactly removing the sensibility altogether, leaves the brain in a confused condition. That is the least serious of the class of case I am talking of; and that is the kind of case of which you frequently hear a great deal, as I have said, after apparent recovery.

Although this may seem odd at first sight, it is really very simple if you analyze the matter. A patient who, after an injury to the head, remains unconscious for a considerable period is certain to be considered to be in a very serious and critical condition, and therefore gets all the care he requires. On the other hand, a patient whose unconsciousness lasts for only a few seconds or minutes or who may, perhaps, not have been really unconscious at all, recovers so quickly that he himself, in the first place, looks upon his accident as a very trivial affair, and, in the second place, the practitioner whom he possibly consults a little later may be inclined to do the same. Thus, a person who receives a slight concussion is somewhat liable to have no immediate treatment at all; and that is the serious point about these particular cases. A patient who has had a slight injury of the kind we are discussing may apparently recover

perfectly at first; at the end of a quarter of an hour or an hour he may feel quite himself again, and, supposing he has been hunting, he may go on to the end of the day fairly comfortably; the following day also he may continue to feel well, but more commonly he feels out of sorts—a little giddy, perhaps, when he gets up in the morning; and he may have a little pain. Please mark the exact situation of the pain in these cases; it is always just at the upper part of the nape of the neck. I mention this particularly, because many persons who have attacks of this sort are apt to attribute this pain about the head to biliousness. Now the pain in the head from an attack of what is called "biliousness"—whatever that may mean—is never at the spot where the head joins the neck; which, on the other hand, is the precise seat of the pain in these "trivial" cases of concussion, supposing that pain is subsequently felt.

Beyond these symptoms, nothing may be felt at all, and so the patient may think that he has quite recovered from the effects of his injury; but the recovery is often a delusion, for frequently at the end of a fortnight, three weeks, or a month, such a man begins to feel that he is not quite himself; he is, in fact, not the man he was. Then, for the first time perhaps, he may seek medical advice; and the practitioner consulted will, if he is a shrewd person, finding certain symptoms, send the patient to bed, and probably keep him there for some weeks.

Now, in the event of any one of you being consulted by such a patient as this, it is clearly of the first importance that you should understand that there are certain symptoms which ought to lead you to the conclusion that the case is more or less serious. It is a very difficult thing, you must understand, especially with the better class of people, to get patients to realize the fact that they are really seriously ill, unless they actually suffer from some gross symptom; and in some of these cases it is a still more difficult matter to get the patient to do what is necessary under the circumstances. The amount of difficulty will depend very much upon yourself, and the amount of personal influence you can bring to bear. There are two points which should lead you to look upon a man's condition as being more or less grave:—(1) the aspect of the face—the expression; (2) the pulse.

The expression to which I refer is present in a good many of these people and can scarcely be described in words, except by saying that it is

unnatural, with a dazed and sometimes almost frightened look.

This peculiarity of aspect, however, may not always exist. The one symptom which invariably exists in all these cases is a characteristic peculiarity of the pulse, which is of the greatest possible importance. The pulse associated with all cases of concussion of the brain, whether severe or slight, has the following distinct characters:—

When first felt by the finger in the usual way it seems fairly good, with indeed nothing much about it to attract attention. It comes up quickly under the finger and seems sufficiently strong; but if the finger presses on it ever so lightly, you will find that it is reduced to absolute nothingness; in point of fact, the slightest possible pressure stops it altogether; it is, in other words, an extraordinarily compressible pulse. It is a pulse which has apparently a great deal of action, but it really has next to no power at all. Indeed, although the expression may seem a somewhat contradictory one, "action without power" would fairly express the kind of pulse you have to deal with in these cases. It is small in calibre, it rises quickly under your finger, and is singularly compressible. In rate it varies from 70 up to 100, dependent on the condition of the patient; but the rate of the pulse is a point of no material importance. So long as a patient who has suffered from any degree of concussion of the brain, however slight it may have been, has a pulse of that kind, you may be absolutely certain that danger still exists, not perhaps directly to life, but at all events there is danger of permanent impairment of health if nothing worse. If you do not quite understand what I say about this peculiarity, please compare the pulse of the next patient you see in the ward with concussion of the brain with your own (provided, of course, you are in an ordinary condition of health); you will at once, I am sure, appreciate the difference between the two. You will find your own to be a roundish, evenly rising pulse, which does not come up to the finger too quickly and is a little difficult to compress; whilst that of the patient is very readily compressible, and possesses the other characteristics I have referred to. Your proper course, then, in these apparently trivial cases of the kind under discussion, is to induce the patient if you can to take at once to his bed, and remain there until the pulse changes its character and reassumes the condition that it

would naturally have in a healthy person. This is one of the most important points, so far as I know, about these cases of concussion of the brain, and it will apply as a diagnostic symptom with certainty in any class of case.

Let me now describe briefly a couple of cases which will show exactly what I wish to insist upon, and will indicate the sort of sequence of events which may, and often does, take place in these trivial cases of concussion of the brain. Such cases are not often met with in the hospital, because, as a rule, it is only the severe class of case which gains admission, the slighter ones come to the out-patient room, then go away, and are lost to us.

Please understand that I do not mean to say that the majority of these cases prove altogether disastrous, or that none of them get well; but what I do mean to say is that every man who has had concussion of the brain at all, however slight or transient the symptoms may have been, has gone through a very critical condition, which if not properly treated at first, may in the end reduce him to the state of a chronic invalid, or possibly something worse.

Case 1. A man, aged 26, had been out hunting. He was of a somewhat heavy build, and weighed 13 st. His head was struck by the branch of a tree under which he passed. He was knocked off his horse, and, so far as I could learn, was unconscious for a few seconds. By the time his horse was caught he was well enough to remount, and finish the day's sport. He thought little more of the matter. When he returned home at night he had a hot bath. He felt a little stiff, but thought it due merely to the hard exercise of the day. At midnight he awoke with some slight headache. The next morning he was a little giddy when he got up, but still he took no notice of his symptoms. Later in the day, however, he felt pain in the back of his neck. He therefore consulted a doctor, who very properly prescribed some Blue Pill, but apparently did not sufficiently impress upon him the importance of complete rest. In fact, two nights afterwards he went to a ball, during which he felt so giddy that he went home to bed. He afterwards resumed his amusements much as usual until at the end of a fortnight he began to feel sick and generally out of sorts. He then came to see me. I had known him for some time, and directly he came into my room I saw that he was not in his natural state. He had that curious expression which I cannot exactly describe to you.

On feeling his pulse I also directly detected in it the existence of the peculiar, thin, apparently strong, but really very weak and extremely compressible character I have described. I told him what I thought was the matter—that he was suffering from the result of the concussion of the brain, and that he had better go home to bed and stop there. Fortunately, I had some influence over him. He went to bed for six weeks, at the end of which his pulse had as nearly as possible regained its natural condition. The case, however, had gone a little too far before he came into my hands, the result being that he did not make a really complete recovery. After a voyage of twelve months on which I sent him, he came back in fairly good health. At the same time he is not, and never will be, the man he was before the accident. He cannot apply himself, as he used to do, to any work he takes in hand, and still there remains a slight suspicion of the peculiar pulse—a condition that will remain, I fear, with him permanently. That is a case in which a man, by neglecting a trivial, or apparently trivial, injury of this kind, to some extent really invalids himself for life. What will happen to him later in connection with his illness I cannot tell. It is possible that nothing further will develop; but it has so far had this effect on him that he has had entirely to change his career, and settle down with the assurance that he will do nothing in life, for his brain is incapable of undertaking work of any useful sort.

Case 2. Another man, aged 28, was thrown from his horse, and fell on the back of his head. That he was unconscious for a short time is certain, as he was seen to be so by a friend. He got up, and in a hazy sort of way managed to catch his horse, and rode for a couple of hours longer. Beyond that time he could not ride because he was so giddy. He, therefore, went home, and obtained medical advice. To the best of my belief he was kept in bed for three days, when, of his own accord, I think, he began to get about. Nothing would induce him, so I was told, to remain longer in bed. When I saw him at the end of six weeks, he was in almost the same condition as the other patient. He lacked the peculiar expression, but had the characteristic pulse. He had very slight pain about his head in the usual spot. The character of pulse alone appeared to me sufficiently conclusive, and I impressed upon him the necessity of doing as the

other patient had done. He laid up in bed for a time, but, unfortunately, my counsels did not prevail with him so well as in the other instance, for he was persuaded by some of his friends that he had recovered, and that it was hardly worth while to remain in bed longer. I saw nothing more of him till some months afterwards, when he came back to me complaining of a somewhat acute pain in the extreme back of his head, in the situation I have described. There was also a certain shrinking from bright light; the characteristic pulse was to be felt, and, I suppose, had existed all the time since I last saw him. He had lost fifteen months altogether, having received no proper treatment. I at once sent him to bed, where he remained for six weeks. His pulse improved to a certain extent, but never recovered in the same degree as it did in the other case; indeed, the patient made but little real progress in the way I should like him to have done. However, he got apparently a good deal better, he subsequently became impatient of restraint, and, therefore, got about again against my advice. To make a long story short, he gradually became less and less capable of doing what he had to do; he soon began to talk nonsense; in a few months time his mind entirely gave way, and he is now in an asylum. These are very good examples to show the course which these cases may take when neglected; I do not say always, but frequently enough to make it worth while to point out the importance of taking them seriously in hand at once, *i.e.*, immediately after the accident. Had these patients gone to bed in the first instance, and remained absolutely quiet *until the peculiarity of the pulse had passed away*, their after-histories would not have been told, for complete recovery would most assuredly have followed.

Such, then, are some of the disasters which may follow upon letting even trivial cases of head injury of this kind take their own way. In the severe cases there can be no question at all about the treatment. If a man is brought to you absolutely unconscious, obviously the only thing to do is to treat him as if he were in a serious condition. In connection with that kind of case, an important point which you will often see illustrated in the hospital will repay consideration. In the course of the treatment of all cases of severe concussion of the brain two questions arise. The first is, When may the patient be allowed to take solid food? and the second is, When may he get up, and

begin to resume his usual occupation? These two questions any cautious surgeon always considers very carefully in connection with the treatment of these cases. I assume that every case of concussion would, in the first instance at all events, be treated by complete rest, very gentle feeding, the diet being limited to beef-tea, milk, and articles of food of that kind, and that no attempt would be made to give solid food until some time had elapsed after the recovery of consciousness. Solid food should indeed never be given until convalescence has undoubtedly commenced. In determining the time at which solid food may be taken, you will have again to be guided by the patient's pulse and expression. As soon as the dazed, somewhat vacant, and occasionally almost frightened look disappears from the face, solid food may be commenced, and the result carefully watched. It is here necessary to understand that this peculiarity of expression will sometimes persist in a greater or less degree for weeks, and possibly for months. The patient may be quite unconscious of it himself, but there it is. At the commencement, the solid food should naturally be of a kind easily digestible, such as fish, etc. It should always be commenced whilst the recumbent position is still maintained. If after solid food of that kind has been taken for two days no ill-effects follow, no giddiness, no increase in pulse rate or temperature, the same food may be continued for another day, the patient being allowed to sit upright in bed. If no ill-effects follow the taking of food with the body in the upright position in bed, the fish, or whatever may have been given, may at the end of two more days (making four in all) be changed to chicken, etc., with ordinary "butchers' meat" to follow, provided that no giddiness, headache, sickness, or acceleration of pulse has occurred. It is hardly, I presume, needful to say that careful attention at the same time should be given to the condition of the bowels.

The next question is, When may the patient get up? That is a very important point. Some persons have an idea that when a man can take solid food without discomfort, he may be allowed to get out of bed and commence to walk about. I have heard statements made to that effect by surgeons of repute. That, however, is not the test by which the propriety of allowing a patient to get about can be decided. To decide this question we have again to rely upon the pulse. You may

perhaps be inclined to think I am possessed by a fad on this pulse question, but I assure you it is not so. You can easily prove the matter for yourselves by watching cases in the wards. *A man should not be allowed to get out of bed to walk about, or even to sit in a chair, until his pulse has assumed its natural character.* When the natural pulse has returned, it is absolutely safe to allow the resumption by degrees of any ordinary business or occupation. There may be weakness, and the knees may be inclined to give way, simply as the result of confinement to bed; but so far as the head and brain are concerned, it will be perfectly safe for the patient to resume his ordinary life. If you will only keep him in bed until the re-establishment of the natural character of the pulse, you will never hear anything more of the concussion of the brain; on the other hand, if you do not exercise this precaution, you must be prepared for some further possible development of the case in the way I have already indicated.

Here is a case in illustration of the preceding remarks.

Case 3. A man, 23 years of age, in the Oxford Ward, bed 16, had a piece of timber fall on his head. He came in suffering rather badly from concussion, and remained unconscious for some time after admission. He had the characteristic pulse of these cases. In about three weeks after his admission the dazed look disappeared, and we began to feed him first with soft solid food and then with the food comprised in the ordinary hospital diet. About that time the question as to his getting up arose one day when I was going round; and I pointed out that I would not allow him to get up yet as the peculiar pulse (which I demonstrated to those of you who were with me at the time) was still appreciable. Two days after that, for some reason which I cannot explain, he was allowed to get up whilst his bed was being made. He got up, stood at the side of the bed, and, I believe, took a few steps about the ward, when he was seized with a curious feeling about his head, and he had a sort of convulsion which caused him to fall back on the bed and become extremely violent. The violence was so great that it was found necessary to have a special nurse with him to restrain his convulsive movements. If that man had not been allowed to get up until his pulse had reassumed its natural condition he certainly would not have had that curious attack. The attack passed off, and the pulse gradually

regained, if not its normal state, at all events something so like it that we could hardly detect the difference between it and the ordinary pulse. We then allowed the man to get up, and, in spite of the attack described, he is now comparatively well.

What the nature of this attack was I cannot exactly tell you. I have seen several similar instances. In one patient I know it was caused by a little hæmorrhage under the cortex which occurred around the seat of an old clot. The nature of the attack, however, is not of consequence for my present purpose. The point that I wish to insist upon is that I have no doubt that it could have been prevented had more care been shown about letting the man stand or move, before he was in a fit condition to do so. That case then illustrates the value of careful observation of the pulse in those very severe cases.

Let me refer to another point of interest, of a different kind altogether. A patient was admitted in the same week as the one just referred to, suffering from severe concussion, but in his case there was a large scalp wound which led to considerable loss of blood. The degree of insensibility was very much the same in the two cases, but although the man with the scalp wound came in after the other, and was certainly as bad if not worse, he recovered consciousness and resumed his natural condition very much sooner. I refer to the two cases together as it allows me to point out a singular clinical fact. All other things being equal, by which I mean, given two patients suffering from the same degree of concussion at the same time, that is, with the same degree of unconsciousness and with all the other concomitant symptoms equal, one having a scalp wound and the other having no wound at all, the patient who has the scalp wound, if it is sufficient to cause a material loss of blood, will recover from the effects of the concussion more quickly than the other. That is a solid clinical fact which you will do well to note, for it is undeniable. It is, of course, interesting, because it is a pretty conclusive indication that the loss of blood in some way or other benefits the patient. A fact like this naturally reminds us of the old-fashioned times when a man who in consequence of a fall or blow upon the head became unconscious was almost, as a matter of course, bled at once to the amount of a good many ounces. It was held by practitioners who thought they knew their business thoroughly, and some

of these old-fashioned people knew their work very well indeed, that a man who was bled became conscious more rapidly than one who was not. That is rather an important point which gives me the opportunity for saying just one word about the treatment of some of these cases of concussion by blood-letting. In these days when bleeding has ceased to be practised, or at least, when venesection is so seldom performed that it is one of the rarest operations in surgery, it seems hardly reasonable to suggest blood-letting in any case of concussion of the brain. Nevertheless I am sure that there are cases of this kind in which blood may be taken with advantage. Here, again, the condition of the pulse will be the guide to the treatment. If in a case of profound unconsciousness from concussion, the pulse is, as occasionally happens, temporarily full or "bounding," moderate bleeding will undoubtedly do good. Some people may tell you that a bounding pulse is never seen in cases of concussion of the brain. It does, however, occur, not very often, but frequently enough to be worth noting, just at the beginning of the reactionary stage. The bounding character is temporary only and soon subsides, if the patient does not die, into the compressible habit upon which I have laid so much stress. In these cases, as a rule, the unconsciousness is very profound, and the patient during this condition of pulse is undoubtedly in much danger; the withdrawal of from six to eight ounces of blood under such circumstances is of great benefit.

In several cases which I have seen of this kind the amount of good produced by a moderate and judicious blood-letting has been incalculable. Although there are some other questions of interest of which I should like to have been able to speak to-day, time does not permit of my doing so. The points, however, upon which I have laid stress are, I am sure from my own experience, well worthy of your careful study for the sake of yourselves as well as for the sake of your future patients.

Night Sweats of Phthisis.—

R. Sulphate of Atropine	0.0005 gram.
Sulphate of Zinc ...	0.12 gram.
Gallic Acid ...	0.12 gram.
Creosote ...	10 gts.

M. Div. in pil. No. v.

Sig.: One pill thrice daily. (*Med. Rec.*)

A CLINICAL LECTURE ON AN INSIDIOUS FORM OF IRITIS.

BY

ROBERT BRUDENELL CARTER, F.R.C.S.,

Consulting Ophthalmic Surgeon to St. George's Hospital.

GENTLEMEN,—The patient whose case I wish to bring under your notice to-day is a single woman, about 50 years of age, of spare habit, and generally of fairly good health. She tells us that her right eye became tender and troublesome about five weeks ago, that she obtained a "lotion" for it from a chemist, and that it improved after a few days. The sight at first was somewhat dim, but became clearer as the tenderness subsided, although it never quite recovered. A few days ago, she had a recurrence of the original pain and tenderness, and also of the dimness of sight; and, as these conditions continue, to-day she comes to us for advice. You will see at a glance that her eye is looking irritated, and that the conjunctiva covering the globe is a little pink, although not in any marked degree red or congested. Vision is at about one-fourth of the normal acuteness; that is to say, she cannot read at a greater distance than ten feet the test types which she ought to read at forty feet. It is always desirable, in examining a case of eye disease, to proceed in an orderly and systematic manner; since in this way we obtain security that nothing shall be overlooked, as it easily may be if we suffer the attention to be at once engrossed by some prominent symptom or complaint.

My own method is to begin by ascertaining the state of vision, first in the right eye and then in the left, so as to obtain this essential element of diagnosis before the eye is either handled or exposed to artificial light. Putting on a trial spectacle frame, and covering the left eye with a disc of ground glass, we obtain the result I have mentioned; while, by transferring the ground glass to the affected eye, we discover that the vision of the left is normal. The patient believes that her two eyes were equally good prior to the first appearance of the symptoms of which she complains; and, although her belief is not evidence, we may perhaps assume that it is well-founded. We must not forget, however, that

patients may discover by a sort of accident, as by closing one eye, a defect of sight in the other of which they were previously unconscious, but which can be ascertained, perhaps by the ophthalmoscope, to depend upon changes of very old date; and cases of this kind are sometimes presented to us in connection with claims against railway companies or other persons for injuries said to have been recently received. At the cost, therefore, of a slight digression, I will mention a test by which recent impairment of vision in one eye may often be distinguished from impairment which has been of considerable duration.

The power of closing both eyes at once is possessed by mankind from the earliest period of existence; but the power of closing one eye without closing the other is one which has to be acquired, like many other muscular movements, by practice. Many people close one eye in taking aim with a gun, or in looking at a very near object (which would otherwise be seen double), or in using a microscope or a telescope. A carpenter closes one eye in order to look down the edge of a plank with the other, and so on of many occupations. Speaking generally, it is found that about 95 per cent. of normal sighted men acquire, almost or quite unconsciously, the power to close either eye independently of its fellow, and to do so without effort or grimace. In normal sighted women, the proportion of those who acquire this power is smaller, and is said not to be more than about 75 per cent., female occupations producing less demand for the accomplishment. Now, a person with one defective eye may often want to close it, and, as it were, to get its imperfect vision out of the way; but such a person would never want to close the good eye, and would never be likely to cultivate the knack of doing so. The orbicularis muscle of the good eye would never have opportunities of independent practice, and hence would never acquire a habit which can be acquired by practice alone. It follows that, in any doubtful case, you may often obtain a clue to the truth by telling the subject to look straight at you, and to close the affected eye. If he can do this easily, tell him next to close the sound eye. If he is able to close either eye with equal facility, you may assume that the defective sight of one, if real, is due to some recent disease or injury. If he cannot close the good eye, or if he can only close it by the aid of struggle or grimace, you will have strong presumptive evidence

that his alleged defect of vision is not only real, but also that it is of long standing.

To return to our patient. Placing her with her back to the window, I stand facing her at about two feet distance, and direct her to cover her left eye with her hand, and then, with her right, to look steadily at my left eye, and not to lose sight of it. I then hold up both my hands, on a horizontal line, one on each side of my face, and in a position midway between her face and my own, and ask whether, while still looking at my eye, she can see them both. She can, and I slowly separate them more and more, with the result of ascertaining that she can see them at as great a distance from the centre as I can myself, so that her field of vision is co-extensive with my own. This shows that there is no contraction of the field, and prepares me for the fact, next to be ascertained, that her right eyeball is of approximately normal tension. Making her look down, I place the tip of one index finger as far back as I can upon the upper lid, under the margin of the orbit, and with this fix and steady the eye; while by the tip of the other index, placed by the side of the first, I feel that the eye can be dimpled by very moderate pressure. In the course of this examination I find that the eye is distinctly tender to the touch.

Proceeding to careful inspection of the affected organ, the first thing noticeable, in addition to the slightly increased vascularity of the ocular conjunctiva, is that the iris is somewhat dull of aspect, and that the pupil is small, rather smaller than that of the sound eye. The irides are hazel, with spots of black pigment; and, without there being any great change, neither the edges of the pigment spots, nor the fibres of the iris, are quite as well defined as they ought to be. The pupil is not only small, but it is also of greyish colour as compared with its fellow. The sound eye being covered, and the diseased eye alternately shaded and exposed to daylight, it is found that the pupil does not vary under these differing conditions; and then, in order to complete the diagnosis, I place within the conjunctival sac two wafers, one containing cocaine and the other sulphate of atropine, and put the patient aside for a quarter of an hour. At the end of that time, we find two small areas of pupillary dilatation, separated by an intervening fixed point; the rest of the pupillary margin remaining stationary and adherent. I then examine by focal illumination, that is to say, by

throwing a convergent pencil of lamp-light upon the eye by means of a convex lens, while the surface thus illuminated is magnified by another. It instantly becomes apparent that the greyness of the pupil is due to a delicate film of effused lymph which covers the centre of the lens, and that the areas of dilatation are clear and black by comparison. Where each of these areas joins the film, the edge of the latter is plainly discernible.

We have, then, to deal with iritis, with inflammation of the iris, in a form so insidious that it might easily escape the notice even of a moderately careful observer, and yet in one which is capable (and the more so by reason of its insidiousness), of leading to the complete destruction of sight. Only two small apertures, corresponding to the two areas of dilatation, remain to afford channels by which fluid behind the iris may obtain access to the anterior chamber of the eye; and this amount of mischief has been accomplished, presumably, by two attacks of apparently only slight inflammation, following one after another at an interval of about a month. They have deposited a film of lymph upon the lens, and to the edges of this film the whole of the pupillary margin, except for the two small apertures, has become adherent. A third attack would be likely to close the apertures, and to reinforce and strengthen the film; and then the iris, with its occluded pupil, would form an impermeable barrier between the fluids and structures which were respectively in front of and behind it. The fluid secreted by the ciliary processes would have no way of reaching the anterior chamber, from which, in the healthy state, it finds egress at the so-called filtration area, the angle, that is, which is formed between the posterior surface of the cornea and the anterior surface of the iris, and in which the venous plexus contained in the canal of Schlemm is separated from the anterior chamber only by a thin layer of comparatively pervious tissue. The ciliary fluid, confined behind the iris, would exert a constantly increasing pressure upon the nervous and vascular structures of the deeper parts of the eye; upon the ciliary nerves and blood-vessels and upon the retina; and this pressure, which might be described as "secondary" glaucoma, would, unless speedily and effectually relieved, entail complete and irremediable loss of vision.

What is to be done to prevent such a disastrous conclusion of the case? Iritis owes its importance mainly to the fact that, in the normal state of the

pupil, the margin of this opening lies in direct contact with the anterior surface of the capsule of the lens, so that the smallest effusion of adhesive lymph causes the two structures to adhere together. In consequence of the convexity of the lens, its peripheral portions lie in a deeper plane than its centre; and hence, when the pupil is fully dilated, the contact of its margin with the lens surface ceases, and the two structures are separated by a film of aqueous humour, of sufficient thickness to prevent adhesions from being formed. When the pupil is contracted, adhesions form quickly, and cannot always be afterwards overcome; but when the pupil is dilated, any lymph which may be poured out as a product of inflammation will mingle with the aqueous humour, and will do no further mischief than to produce a temporary turbidity. In the early stage of iritis, if the pupil can be fully dilated and maintained in dilatation, the inflammation will die out harmlessly; and hence, in this early stage, it frequently happens that no other treatment than the application of atropine is required. On the other hand, when the effusion has had time to acquire firmness, and when the dilatator muscle is weakened by inflammation, it is very possible that the adhesions may resist atropine. Our sheet anchor then is mercury.

In the instance before us, the adhesions probably date, some of them at least, from a time five weeks ago; and I think it would be unsafe to trust to atropine alone. The woman receives a bottle of drops, containing one grain of neutral atropine sulphate, and double that quantity of cocaine hydrochlorate, to two drachms of distilled water, and she is instructed to make three applications of this liquid daily, each application being threefold. A drop is to be inserted within the lower lid, in five minutes another, in another five minutes a third, and this is to be considered as one application. She also receives pills, each containing two grains of blue pill, with a quarter of a grain of opium, and is told to take one of them three times a day. She cannot come into the hospital, but is able to take some care of herself at home; so she is forbidden to read or to do needlework, or in any way to tax the sound eye, and is cautioned against exposure to cold winds, to strong light, or to the heat and glare of a fire. If she were a cook, I should insist on her taking a complete holiday. She is to wear a small shade, and is to protect the eye on leaving home by the addition of a pad of cotton wool.

(Three days later.) Our patient with iritis returns to-day in an entirely satisfactory condition. The pinkness of the affected eye, as well as the pain and tenderness, have much diminished, the adhesions have all yielded, and the pupil is fully dilated and circular. The sight is still defective, and focal illumination still shows the presence of the film of lymph, although it is less conspicuous than before. On looking through the dilated pupil with the ophthalmoscope, with a strong magnifier (16 dioptries) behind the mirror, there is an appearance of a deeper turbidity, probably from cell proliferation in the anterior layers of the vitreous. The gums show no trace of mercurial action. The patient may now apply the drops night and morning, each time once only, and may take one of the pills every night. By the end of another week she will probably be practically well.

The chief lesson to be derived from the case is the very important one that iritis may occur, and may inflict serious injury, without presenting symptoms of sufficient severity to call for very much notice, or, in other words, without the acute pain, or the superficial redness, or the great impairment of sight, which are often described in text-books, and which are certainly often present in what may be called typical cases. It is, therefore, necessary to examine even slight examples of inflammation of the eye with great care, and always to recollect how much mischief may sometimes lurk under a very small amount of outside show. One of the chief dangers of an error depends upon the fact that iritis, if mistaken for mere surface inflammation or conjunctivitis, and treated by any of the so-called astringents, such as zinc lotion, is liable to be roused into greatly increased activity. It is necessary, before prescribing any local application, to inspect the state of the pupil with due care, and if there be the slightest doubt, to use atropine as a means of removing it. If the chemist's "lotion" in this case had contained atropine, the patient would probably have been well within a few hours of her first visit to him.

Assuming that the result had been less favourable, and that some of the adhesions had resisted, as they often will resist, the atropine and the mercury, the eye would, after apparent recovery, have been left exposed to recurrences of the disease. These recurrences seem to be provoked by the presence of adhesions, and by the hindrances which they offer to the physiological variations in

the diameter of the pupil. If adhesions are left after a first attack, it is not desirable to do more than keep the patient under observation; but the habit of recurrence, if once established, will be likely to lead ultimately to destruction of the eye, and it is usually best to anticipate such a consequence by the performance of iridectomy at a time when things are comparatively quiet. If a considerable piece of the iris be excised, and the continuity of its circle broken, the irritating effect of any remaining adhesions will usually be found to disappear. Recurrences of inflammation may occur from time to time, but they will mostly be separated by increasing intervals, and will present characters of diminishing severity. When this is the course of events, the habit will ultimately be broken, and a thoroughly good and useful eye may be retained.

THERAPEUTICAL NOTES AND FORMULÆ.

Typhoid Fever.—Under the title of "Can Typhoid Fever be Aborted?" Dr. J. E. Woodbridge has lately contributed to an American contemporary a series of articles on what he claims as a new treatment of his own. We trust he will forgive us if we express a little hesitation in accepting his figures as literally correct, without any explanation, when he says:

"Thus far in my private practice I have had no death from typhoid fever for twelve years. I have been able to abort two or three cases when first seen on the tenth day, and *all* who came under my care on or before the eighth day of sickness. It may not be possible to abort every case, beginning on the eighth day, nor is it necessary; for when the profession and the people understand that typhoid fever can invariably be cured when proper treatment is instituted at a sufficiently early period, the physician will no longer wait until his patient is covered with petechiæ, or has had one or more hæmorrhages before making a diagnosis or beginning treatment."

However, as the "Journal of the American Medical Association," corresponding with our own "British Medical Journal," has published his results, apparently satisfied with the *bona fides* of his successes, we feel it our duty to give our readers the outline of the treatment that they may, if so disposed, give it a fair trial. In our own

opinion the drugs in the quantities prescribed are certainly harmless, if they will not do all that Dr. Woodbridge claims for them. His prescriptions are as follows:

No. 1.

R	Podophyllin	gr.j
	Hydrarg. Subchlor.	3j
	Guaiacol Carb.	3vj
	Thymol	3v
	Menthol	3j
	Sacch. Alb.	3ij

M. Ft. pulv.

Sig.: gr.j or ij every half-hour.

No. 2 as a diuretic.

R	Potass. Acet.	3j
	Sp. Æth. Nit.	3ss
	Aq.	ad 3iv

M. Ft. mist.

Sig.: 3j every half-hour in water or lemonade.

No. 3.

R	Eucalyptol	3ss
	Spir. Rectif.	3j
	Guaiacol	3ij
	Aq.	ad 3iv

M. Ft. mist.

Sig.: 3ss every three or four hours.

His method of applying them varies somewhat, so we quote his own words:

"During the first two days the patient (case 51) took about 60 minims of Eucalyptol, 15 minims of Guaiacol, 30 minims of Turpentine, and about 15 grains of No. 1 powder. During the following three days, he took 2½ minims Guaiacol and 5 minims Eucalyptol every three or four hours all of the time, and part of the time double that quantity with an occasional 10 drop dose of Turpentine added; and in addition small doses of Quinine continuously every three hours; occasionally a 2½ grain tablet of Dover's powder. Nearly every day during his sickness, and sometimes twice a day, he had rubbed on his abdomen a mixture of Eucalyptol and Guaiacol, with the addition, sometimes, of Turpentine. His kidneys failing to respond to the Turpentine, there was given him No. 2 as a diuretic; and when the bowels became constipated 'Glycerin suppositories' were used.

"Case 52.—W. H. took R. No. 1 two days in about ½ grain doses every thirty minutes. For two days, No. 3; for one day, No. 1; No. 3, two days; No. 1, one day; then No. 3 until the temperature had been subnormal two days.

"Case 55.—Diagnosis, typhoid fever. Name, T. M. Date of admission, Feb. 6, 1894. Diet: Ate beefsteak on eighth day, beefsteak and blanc mange on ninth day, and whatever food he desired thereafter.

"He took No. 1 two days; Eucalyptol and Guaiacol, two days; No. 1, one day; Eucalyptol and Guaiacol in one mixture, and Thymol in another to the present time (Feb. 12, 1894).

"This was practically the course pursued with all the cases reported."

Aerated Milk.—The idea of charging milk with gas (CO₂), and introducing it into medical practice, originated with Professor Botkin, but the more-extended use of it is due to the impulse given it by Prof. Pasternacki, who treated patients extensively by aerated milk in 1890, at Kislovotzck, and became convinced of its value. In his opinion aerated milk may be used in preference to ordinary cow's milk, as it is more palatable to sick as well as healthy persons, especially during the heat of summer, is a refreshing drink, and quenches the thirst. It may also be used as a substitute for kefir and koumiss, when the latter cannot be given or is not well borne by patients. It is especially useful in cases of feeble digestion, and patients limited to substances that require no great effort of the digestive organs. Favourable results have been obtained by the use of alkaline waters with aerated milk, enabling patients to endure the treatment.

Borysowski states that the best and cheapest method of aerating milk is by liquid CO₂, which, in St. Petersburg, is made in special factories and distributed in iron bottles, under pressure of about sixty atmospheres. He gave preference to the apparatus constructed on the principle of Wulf,—a three-necked bottle used by Pasternacki. The aeration lasts about forty minutes under pressure of 1½ to 2 atmospheres. The apparatus used for aerating larger quantities (8 or 9 litres—8 to 9 quarts) is made of a metallic cube, in the top of which are four leaden tubes, one reaching to the bottom, connected with the bottle containing liquid carbonic acid; the second, also reaching to the bottom, for pouring the milk into the glass when ready; the third for letting out the air, and the fourth for the manometer. In order to enable patients to aerate milk at home the author

constructed an apparatus consisting of two tubes soldered together and passed through a well-fitting cork, a long tube for CO₂, and a short one for the escape of air. Curds, albumen, albuminose, and peptone do not change qualitatively by aëration. Microscopical examination of aërated milk proves that the corpuscles diminish in size without changing their form, also that small curds may be observed.

(*Inaugural Dissertation*, St. Petersburg, 1893.)

A Catarrh Snuff.—The following is recommended by a writer in a French journal as a useful remedy for acute coryza:

R	Salol	gr.xv
	Salicylic Acid	gr.ijj
	Boric Acid	ʒj
	Tannin	ʒiiss

Sig.: To be used as a snuff in the early stage of an acute rhinitis. (*Med. Rec.*)

Bromidrosis.—Internally give ℥viiij Tinct. Belladon. in ʒss Fluid Extract Ergot, three times a day; externally, use on the body the following powder several times daily:

R	Salicylic Acid	ʒijj
	Boric Acid	ʒijj
	Zinc Oxide	ʒss
	Powdered Starch	ʒv
	Ottar of Roses	℥xx

(*Med. Rec.*)

Bronchitis with difficulty in coughing up secretions.—

R	Ol. Terebinthin.	ʒij to ʒijj
	Mucil. Acaciæ q. s.			
	Aq. Cinnamomi	ʒj
	Aquæ q. s.	...	ad	ʒvj

M. Sig.: A tablespoonful in a little water every four hours.

The Care of the Teeth in Children.—Dr. A. E. Baldwin, of Chicago, draws attention to the necessity of preserving the temporary teeth, especially the molars, which are not usually replaced till the tenth or twelfth year. The necessity is proved by the well-known but too little regarded fact, that the child needs well masticated food, in greater relative abundance than the adult, to provide for rapid growth and development over and above mere waste and repair. If the temporary teeth are neglected they soon become sensitive, and then the

child will not masticate properly, and soon acquires the very bad habit of bolting its food. Dr. Baldwin goes on to say that if room is absolutely necessary for a permanent tooth to come through, only the *one* temporary one should be removed, as the jaw by its expansion gives the required extra additional space.—(*Journ. Amer. Med. Assoc.*)

REVIEW.

Medical Annual for 1894. (Wright & Co., Bristol.)

Published at 7s. 6d.

Annual epitomes of the progress of our art are becoming more numerous, and one may say more necessary with each year; the one before us certainly, we think, ranks very high amongst them, if it be not actually the best. In general plan it must naturally fall very much into the groove of all such productions; but Messrs. Wright certainly deserve praise for the lavish way in which they have allowed illustrations to be introduced, and for the high degree of excellence which these have reached. We can scarcely find a single woodcut which is not perfectly clear and very distinct in outline, while the full-page coloured plates are as nearly perfect as art can make them, though we regret that several of both forms of illustration have not the description printed in the margin—an improvement we suggest for future volumes. We admire also the international character of the work: America, Germany, France all contribute their quota, as well as our own provincial schools. W. Bull, Prof. H. Hare, Mayo Robson, R. Saundby, Prof. Unna, Sir A. Garrod, E. Fenwick, Calcott Fox, are names which guarantee that their contributions are well worth reading, and their selections from the works of others judicious. The addition of lists of lunatic asylums, homes for inebriates, training institutions, hydropathic establishments, and books of the year, etc., makes the Annual complete. We can scarcely understand how such an excellent work is produced at the low price at which it is offered.

PUBLISHER'S ANNOUNCEMENT.

As the "CLINICAL JOURNAL" has a wide circulation amongst Practitioners and Students, advertisements can be received for Assistants, Partnerships, Transfers of Practices, etc. Terms, 2s. 6d. for each insertion of four lines or under, and 6d. for each extra line.

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THE CLINICAL JOURNAL.

WEDNESDAY, APRIL 11, 1894.

A CLINICAL LECTURE

ON

A Fatal Case of Raynaud's Disease

In a Girl having Mitral and Tricuspid Stenosis, Pericarditis, Acute Cardiac Dilatation, Pneumonia, Embolism of the Brachial Artery, and Atrophy of the Muscles of Feet.

Delivered at Guy's Hospital, Feb. 10, 1894, by
W. HALE WHITE, M.D.,
Physician to the Hospital.

GENTLEMEN,—The case we take to-day is that of Sarah Ann Hodson, æt. 16, who came into Miriam Ward on January 19th, 1894, for dry gangrene of the toes of both feet and of the right hand and forearm.

Her report says that the patient is an orphan, who has been much neglected. She has never had rheumatism. For the last three years she has had palpitation and shortness of breath, and has been treated for heart disease as an out-patient at King's College Hospital. She said that during this time her hands and feet have often got very cold and sometimes blue. She has had great difficulty in getting them warm again even before a fire. Occasionally in the summer she has suffered from this. There is no account of her ever having been lame.

History of present disease. On January 13th she felt pain in her feet and legs, and noticed that the toes of both feet were turning blue. On the 15th the tips of the fingers of the right hand became blue. They gradually got darker, and the discoloration spread upwards till it reached the point at which it stood on admission. The date of onset of the present febrile illness is doubtful.

Condition on admission. She is extremely dirty, and badly cared for. Pulse 116; respirations 40; temperature 101.2°.

Her right hand and forearm up to within 3½ inches of the olecranon are gangrenous. The fingers are quite dried up, cold, of a dark mahogany colour, and horny looking. The veins show up against this dark background. The forearm

is not so dry, and is of a blue-black colour. The upper limit of the gangrene is marked by a red line. No pulsation can be felt in the right brachial artery, but the third part of the axillary can be felt beating.

All the toes are gangrenous. On the right foot the gangrene extends about an inch beyond the base of the clefts between the toes. On the left foot it extends no further than the clefts. The tips of all the four inner toes on the right foot are dry, as are the tips of the three inner toes on the left. The dorsalis pedis and posterior tibial arteries on both sides pulsate well. The colour of the gangrene of the toes is the same as that of the arm. Its upper limit is, however, less sharply defined, and on the dorsum of the right foot there are two bullæ in the gangrenous area. There is nothing abnormal about the right hand. No scars are to be seen on the tips of any of the fingers or toes.

Heart. Impulse diffuse. Apex in the seventh space just outside the nipple line. Presystolic and systolic thrills can be felt over the apex. A presystolic murmur and a high pitched systolic murmur are audible here; the latter is conducted round into the axilla, and is very loud at the back. The second sound is accentuated. A slight pericardial rub can be heard.

Lungs. Right side healthy. On the left side there is a pleuritic rub in the axilla. At the base of the left lung behind, and extending up to angle of scapula, dulness, consonant rales, and loud bronchial breathing are present. The sputum is rusty.

Urine. Healthy.

Spleen. Neither enlarged nor tender.

Eyes. Optic discs and retinal arteries normal. She is menstruating.

The diagnosis made was that the patient had chronic mitral obstruction and regurgitation. That she had acute pneumonia and pleurisy on the left side with pericarditis, and that this acute illness in a girl predisposed to Raynaud's disease had caused the gangrenous form of it to develop in the toes. The gangrene of the arm was regarded as embolic, secondary to the condition of the heart. Whether or not there was malignant endocarditis was left an open question. As six hours after ad-

mission the apex of the heart had gone two inches further out, it was thought that the pneumonia and pericarditis were inducing acute cardiac dilatation.

Turning again to the report, we find she is ordered Tincture of Digitalis, $\mathfrak{M}\text{x}$; Tincture of Nux Vomica, $\mathfrak{M}\text{x}$; Chloroform Water, $\mathfrak{z}\text{j}$, every four hours. The gangrenous parts are to be wrapped up in cotton wool. She is to be kept warm with hot water bottles, and to be fed upon milk and eggs.

January 20th. The temperature has remained about 103° ; respirations about 40; pulse 160. The physical signs are the same, except that the apex of the heart has moved still further out. The medicine is henceforth to be taken every two hours. She is to have 2 oz. of brandy a day, and a subcutaneous injection of $\frac{1}{4}$ gr. of Morphine every evening, because of the pain in the gangrenous parts, and because of cardiac distress and sleeplessness.

January 21st. The temperature, pulse, respiration, and physical signs are much the same. The gangrene of the arm and toes is unaltered. On the left gluteal fold is a dusky red patch three inches in diameter, bluish in the centre, and yellow at the circumference like a bruise. This spreads across the middle line of the sacrum to the right side, where there is a similar patch. The symmetry of this makes us think it part of the Raynaud's disease. The apex of the heart is beating just posterior to the mid-axillary line, and the diastolic murmur cannot be heard. As she has troublesome diarrhoea, Compound Catechu Powder is ordered, and as everything else fails to quiet her and to relieve her pain, she is given $\frac{1}{4}$ gr. of Morphine subcutaneously regularly night and morning.

January 24th. Since the last note the patient has improved. Temperature to-day 99.8° . The blue black colour of the back part of the gangrenous forearm is lighter, and so also are the fourth and fifth toes of the left foot and the patch on the gluteal region. The pulsation in the various arteries is the same as on admission. The apex of the heart is returning to its normal position, being now only one inch outside the nipple line. The pericardial rub has disappeared. The impulse is less diffuse. The diastolic murmur has returned, and is now mid-diastolic, the systolic murmur is unaltered. The signs of pneumonia are clearing up.

January 27th. The patient is better, her temperature is normal, the heart's apex is in the normal position, the murmurs are the same as on

January 24. There is only a little bronchial breathing at the base of left lung. The condition of the gangrene is unchanged. The patient's chief complaint is of great cardiac distress.

January 31st. Since the last note the temperature has been rising, and yesterday was 102.6° . As the previous improvement renders it unlikely that the endocarditis is malignant, as the pneumonia has cleared up, and as the upper part of the gangrenous area of the forearm is becoming moist, the arm was to-day amputated through its middle, for it was thought possible that the patient's temperature was due to septic absorption.

February 3rd. The operation did not make the patient better or worse, the wound always looked healthy. The cardiac distress increased, and she died to-day.

Now, Gentlemen, when she came in, two criticisms were urged against the original diagnosis. It was in the first place suggested that the patient had malignant endocarditis with consequently a considerable tendency to embolism, and that therefore the gangrene of the right arm and both feet was all embolic. But the reasons against this were very strong. In the first place, as the posterior tibial and dorsalis pedis arteries on both sides were beating well and forcibly, to produce gangrene of the toes there must have been an embolus in the dorsal pedis just before it gives off its communicating branch, and in the external plantar artery in each foot, for, owing to the free communication between these arteries by means of the communicating artery, embolism of one alone will not cause gangrene of the toes. Just think of the chances against such a distribution as a pair of emboli in both feet. Also it is very doubtful whether such emboli would explain the extension of the gangrene on the right foot upwards beyond the toes. The commonest seat for an embolus is the spleen, but there was no evidence of this during life, and the one found at the autopsy was small and unimportant. Then, too, the retinal arteries are a frequent seat of emboli, but they were free in this case; in fact, there was no reason to suspect embolism of any internal structures. It seemed, therefore, that not only did embolism fail to explain the gangrene of the toes, but that there was no wide showering of emboli about the body, such as would have been expected if there were four emboli in the feet, although, from the lack of pulsation in the right brachial artery, we believed that an embolus was

plugging it at its upper end. The subsequent history supported the view here urged for the fall of temperature, and contraction up of the dilated heart rendered malignant endocarditis, which alone would have explained an extensive showering of emboli, unlikely; and therefore we concluded that what had happened was that some non-infective ante-mortem clot accidentally detached, either from a vegetation, or from its seat of formation in some recess on the left side of the heart, had become lodged in the right brachial artery, and had thus caused the gangrene of the hand and forearm, but that the gangrene of the toes was not due to embolism.

Other critics suggested that the case was one of acute arteritis. The unfortunate part of this suggestion is that it is extremely doubtful if such a disease exists. Wilks and Moxon say they know nothing of it. They say, indeed, that cases of chronic arteritis are described, but with our patient the onset was certainly acute. Dr. Hadden has recorded one case of arteritis, and Mr. Pearce Gould has recorded two, and he gives references to others, but Mr. Gould is careful to point out that it is characteristic of the disease that there is "slow mummification of the part." I think, therefore, we may safely say that the patient had not arteritis.

Now, let us turn to the account of the post-mortem.

Post-mortem examination.—Here are the organs before you, and most of the points I can now illustrate. On the left side of the chest there were 22 oz. of turbid fluid, with flaky lymph in it. The lung is tough. On the right side there were 2 oz. of fluid, and there is much recent bronchopneumonia. The pulmonary artery is slightly atheromatous.

There are a few adhesions about the pericardium of such an age that they would correspond to the patient's pericarditis. There are some older adhesions on the front. The cardiac muscle is in many places very fatty and degenerate. The mitral orifice measures only $2\frac{1}{2}$ inches in circumference. The valve is much thickened, and it is a typical button-hole mitral with shortened chordæ tendinæ. The aortic valves are competent, but are fringed with a few very minute vegetations. The cavity of the left ventricle is normal, but the left auricle is much dilated and hypertrophied, and its endocardium is very thick. The tricuspid orifice is rather constricted, measuring only $3\frac{1}{4}$ inches

in circumference, and the valve is somewhat thick. The heart weighs 14 oz.

Kidneys. There is some early tubal change. They are congested, and weigh 9 oz.

Spleen. There is one small infarct of some date. Weight 5 oz.

Liver. Normal. Weight 45 oz.

After the amputation the arm was dissected, and the muscles are seen to be healthy, and those in the stump are also healthy. Here, as you can see, a non-septic embolus is firmly fixed in the commencement of the right brachial artery, and a thrombus extends downwards for about an inch and a half from the embolus. The rest of the artery and the axillary are empty. The arterial walls are healthy. The muscles of the left hand are healthy. The posterior tibial and dorsalis pedis arteries in both feet are healthy and contain no emboli. The muscles of the legs are healthy. The muscles of the soles of both the feet, with the exception of the accessorius on one side and a few of the interossei on the other, are pale and in a state of degeneration.

Microscopical examination of the plantar and posterior tibial nerves shows that there is no well-marked neuritis, the greater number of the nerve fibres are quite healthy, and there are none which could be called certainly diseased, but here and there there is slight evidence of inflammation, as shown by an excessive exudation of leucocytes. We may undoubtedly say there is no primary peripheral neuritis, and I think the proximity of the gangrene will explain all that we have found. Microscopical examination of the degenerate muscles of the soles of the feet shows that the greater number of the muscle fibres are replaced by fibrous tissue, but that the few that remain are for the most part healthy. There is much fat between the bundles of fibres.

I think the autopsy proves that the original diagnosis was correct. You can see for yourselves that there is mitral constriction, that with that valve there must have been mitral regurgitation, you will notice the dilatation and hypertrophy of the left auricle induced by the mitral constriction and regurgitation. The extremely bad condition of the cardiac muscle explains the ease with which, when the pneumonia and pericarditis supervened, the heart dilated. In one other case of severe illness occurring in an alcoholic subject suffering from heart disease, I remember seeing the heart dilate as rapidly as in this patient. You will notice here that in a few days we

actually watched the apex of the heart move out some two or three inches, and return again three or four inches to its normal position. The heart may dilate acutely in any specific fever, especially rheumatic and scarlet fevers, even if there has been no previous cardiac disease. The remains of the pericarditis we discovered during life were found at the post-mortem, and no doubt the pericarditis aided the dilatation. There was no malignant endocarditis. You will notice the tricuspid valve was a little constricted, and this will remind you that this association of tricuspid with mitral stenosis is fairly often met with; why it should occur is not known, it certainly is not due to congenital disease as are most of the diseases on the right side. Tricuspid stenosis is much more frequent in women than in men; it is usually a chronic condition, and commonly much less advanced than the corresponding mitral constriction. This case illustrates all these points.

In the lungs you will notice that the pneumonia on the left side has cleared up, but that there is much pleurisy which might well correspond to the pleurisy which existed on admission. The broncho-pneumonia on the right side occurred shortly before death, as probably also did the left-sided pleuritic effusion, because two days before she died the left base was fairly resonant.

The embolus was found, as we thought, blocking the upper part of the brachial artery. You will notice that it is non-septic, and is producing no inflammation of the arterial wall, and has therefore not been derived from malignant endocarditis, but is due to an accidental detachment of some ante-mortem clot from the heart. The embolus in the spleen doubtless had the same origin as that in the brachial. As we prophesied, the arteries of the feet are healthy.

A little early tubal change, such as is present in the kidneys, may be seen in persons who have suffered from acute pneumonia, and very likely this patient's high temperature had something to do with it.

Both to the naked eye and to microscopical examination the muscles of the soles of the feet were in exactly the condition that would be left after an attack of anterior poliomyelitis; and as the nerves were almost healthy, it seems to me most probable that at some former period our patient had had an attack of this disease, affecting such of the cells of her anterior cornua as are connected with the plantar muscles. Atrophy of

these muscles would not, when boots or shoes are worn, cause any easily noticeable difficulty of walking.

Now we have left to explain the gangrene of this patient's feet, which was, I think, due to Raynaud's disease. We had better, therefore, try and get a clear idea exactly what is meant by this.

Raynaud's disease in its typical form is a malady in which, from time to time, the fingers, toes, and occasionally the nose and ears, or rarely other parts, become symmetrically painful, cold, and livid. It is essentially paroxysmal, there are long intervals during which there is nothing the matter with the parts which at other times are affected with recurrent paroxysmal attacks of the malady. The attacks vary much in severity in the same patient and among different patients. Sometimes they are little more than the lividity and coldness of the hands that all of us are subject to in cold weather, sometimes they pass on to gangrene of the affected parts. Let us now run over the points of Raynaud's disease and see if our case showed them.

1. *The symmetry of the affection.* This is very characteristic, although not essential. It is rarely absolute, but still it is very exceptional for one hand to be affected and the other free. In our case the fact that the toes on both feet were implicated was very suggestive of Raynaud's disease, and the circumstance that the arm on one side only was affected, pointed to the fact which the absence of pulsation in the brachial confirmed, that the condition of the arm was not due to Raynaud's disease. The symmetry of the livid patch in the gluteal region suggested that this was related to the Raynaud's disease.

2. *The state of the parts.* In the mildest degree of the malady the affected part all at once becomes pale and cold. The patient says it feels dead. Its cutaneous sensibility is lowered, it feels cold and is cold, as may be shown by the thermometer, and drops of sweat may appear. This condition may last a few minutes or a few hours, and in the period of reaction the area implicated flushes, and is red and painful.

In the next degree of severity the parts are of a cyanotic blue, they may present all depths of colour from a bluish white to slate coloured or black. The colour may be uniform or there may be much mottling of the blue with dusky red patches. I show you drawings, from a patient you may remember in Mary Ward, giving a very good

idea of the condition of things. The pain,—often shooting and burning,—and the numbness cause the patient to complain much. As the attack gradually passes off the lividity disappears, and a vermillion border appears at the edge of the affected parts. The degree of severity here described is very usual and typical, and is called by Raynaud one of local asphyxia. Sometimes the fingers or toes are a little swollen, but there is never the clubbing seen in the lividity associated with disease of the chest.

In the next degree of severity the local asphyxia does not pass away, and gangrene supervenes. The extent of this is variable; a very common condition is for only the tips of the fingers or toes to be gangrenous, there being little patches of gangrene on their pulps, a gangrenous ulcer forms here, and leaves a small permanent scar which remains for years after the local asphyxia has passed off, so that these little scars are very typical of previous Raynaud's disease. This drawing shows them well. If a large area, such as several fingers, become gangrenous, the gangrene is usually ultimately dry, the mummified parts assume that dark mahogany colour which is so characteristic, and the veins are then seen as dark lines. Sometimes the nails only drop off, and sometimes bullæ filled with bluish fluid are formed on such part of the gangrene as is not yet dry. In the specimen I show you, taken from our patient, you notice the dry gangrene of the toes and the bullæ on the dorsum of the foot.

3. *The history.* The patient usually tells us that she has often suffered, especially in the winter, from attacks of cold livid hands and feet, and you will remember that our patient said she was so liable to them that they even occurred in the summer. This is very characteristic of Raynaud's disease.

4. *Parts affected.* These are commonly the fingers, toes, also of the nose, and ears, the fingers and toes being very much more common than other parts. In our case it was the toes that were principally affected, but she also illustrates that the lividity rarely may begin on any part of the body, for the symmetrical patch on the gluteal region was probably part of Raynaud's disease, and here is a drawing of a patient who was in Mary Ward six months ago, and who from time to time got livid patches on various parts of the body.

5. *The pain.* The affected parts are very pain-

ful. Raynaud himself says that sometimes the patients howl with the pain, and I see from time to time—I have seen her this week—a patient in whom the pain is fearful; when an attack is on she will not let anyone even touch her hands, although they are but slightly livid. In our patient there was much pain, but not so severe as this.

6. *The exciting cause.* The attacks are, other things being equal, always worse in cold weather. The case I have just referred to gets them every winter, and in our patient the condition came on shortly after very cold weather. Also, the more the patient is out of health the more likely she is to get an attack. No doubt in our case the pneumonia was largely responsible.

7. *The pulse.* The pulse can always be felt in the affected parts or right down to them if they are gangrenous. We always, in our patient, felt that both posterior tibial and both dorsalis pedis arteries supplying the toes affected with Raynaud's disease were pulsating, but we could not feel any pulse in the brachial blocked by an embolus.

8. *The sex.* Almost three quarters of the cases are women. The last five cases I have seen have all been women.

9. *The age.* The patients are usually a few years either side of 20. Four out of the five women just mentioned have been of this age, and our present patient was 16 years old.

10. *Menstruation.* Raynaud himself was inclined to regard stoppage of menstruation as an important cause; but I have often asked about this, and have never been satisfied myself that it is of much importance. You should note that our patient menstruated during her illness, and we constantly wondered that, being so ill, she could live so long. This is of interest, because I fancy that when patients are seriously ill and yet menstruate, it indicates a good prognosis, for it shows that the natural functions of the body are not so impaired as we might expect.

11. *The joints.* You will sometimes observe in patients affected with Raynaud's disease that there is fibrous ankylosis of the terminal phalangeal articulations, and that there are thickenings along the processes of the palmar fascia. These results of the disease may in time clear up. Occasionally there is fluid in the joints.

12. *Association with nervous diseases.* Some of Raynaud's cases were hysterical, some patients I have seen have been so, and we all thought the subject of the present lecture decidedly hysterical.

Several cases have also been described from asylums.

13. *Hæmoglobinuria*. It is a well known fact that hæmoglobinuria and Raynaud's disease may occur in the same patient.

14. *Scleroderma* is sometimes associated with Raynaud's disease.

15. Some reported cases appear to show that occasionally Raynaud's disease is associated with ague and with syphilis.

16. It is also very important to remember that it is sometimes associated with urticaria, with ill-defined livid mottlings of the skin, which come and go, and with a condition of the hands and feet, originally described by Weir Mitchell, and termed by him erythromelalgia, in which there are paroxysmal attacks of redness instead of blueness. This drawing shows the mottlings of the skin seen in the case we had in Mary Ward, and I occasionally see a patient in whom sometimes the attacks are most like Raynaud's disease, and sometimes most like erythromelalgia.

17. *The blood*. It is stated that the blood does not form rouleaux so well as healthy blood, this was noticed in a case which was in Miriam Ward under my care in 1890.

18. Lastly, I should like to direct your attention to the fact that our patient had mitral disease. The patient mentioned above whose attacks are so painful also has mitral constriction. Whether this association is a mere coincidence, I cannot say, but it is curious that two at least of Raynaud's original cases also had organic heart disease.

Pathology. You have only to read an account of Raynaud's disease, or to watch a case to see that one factor at least in it is a constriction of the minute vessels of the affected part. Indeed, Raynaud has published two cases in which paroxysmal constriction of the retinal arteries was actually observed, and it led to paroxysms of amblyopia. We may, therefore, start with the assumption that the local syncope and the local asphyxia of Raynaud's disease are due to spasm of the minute vessels of the affected part. Of course you therefore at once say that surely the seat of the malady must be in the nervous system, and of seven published cases in which the peripheral nerves have been examined, peripheral neuritis was found in five, and you well remember the suggestive fact that local attacks of dead fingers are met with in the subjects of peripheral neuritis. At first sight the degeneration of the

muscles of the soles of the feet met with in our case, made it seem, at the post mortem, probable that we should find the plantar or posterior tibial nerves would be affected with long-standing severe neuritis, in which case the degeneration of the muscles would have been secondary to the neuritis, but we did not find any severe neuritis, that which was present was quite inadequate to explain the condition of the muscles. It may be that some day we shall learn that there is a special significance in this atrophy of the muscles of the soles of the feet met with in our patient, for paralysis of these muscles has been described in one case of erythromelalgia, but for the present it seems to me wiser to regard it as evidence of old anterior poliomyelitis.

But please do not go away with the impression that you know all about Raynaud's disease when you have ascribed it to peripheral neuritis, probably it is due to it in some cases, but not only have we failed to find a marked degree of it in our case, but the two published cases in which it was not found were particularly carefully reported and examined. You will find an account of them in Dr. Barlow's translation of Raynaud's work. Then again there is the possibility that sometimes the neuritis is secondary to the gangrene, and this probably explains the slight neuritis in our case, and also it is well-known that many cases of Raynaud's disease have very few symptoms of peripheral neuritis, and lastly, you must remember that local vaso-motor changes can be produced by disease of other parts of the nervous system. For instance, local dilatation of the vessels of one side of the body occurred in the man who had fits as a result of cerebral syphilis, and who formed the subject of a clinical lecture a month ago, and Dr. Fagge has described a case of malignant disease of the thorax in which some of the nerves going to the arm were pressed upon, and the fingers presented a condition very like Raynaud's disease. Also many of the cases of Raynaud's disease occur in hysterical and insane subjects. Then, too, there is the suggestive fact that our patient may have had anterior poliomyelitis affecting the anterior cornual cells connected with the feet, perhaps this left them in an unstable condition, in some way related to the Raynaud's disease of this patient's feet. So you see the right way of looking at Raynaud's disease is to take a wide view, and to regard it when marked as a symptom indicative of disease affect-

ing some part of the nervous vaso-motor system, shading off, quite imperceptibly, into the cold, dead fingers to which we are all liable. Indeed, were any charlatan to set up as a specialist in Raynaud's disease he would probably find that we all had it. This view makes clear its wide and many relations; for instance, it explains why it may be associated with erythromelalgia, which is a vaso-motor neurosis in which the vessels dilate instead of contracting, and in which, as I have already mentioned, paralysis of the muscles of the feet has been observed, reminding us of the state of the muscles in our case. It renders the association of Raynaud's disease with urticaria and with local irregular mottlings intelligible, and shows that the girl in Mary Ward in whom mottlings came out from time to time in various parts of the body, was really suffering from a variety of Raynaud's disease, and this view also warns us not to necessarily expect to find an organic cause for the disease; we can no more hope always to find an organic explanation for paroxysmal vaso-motor attacks leading to blueness of the fingers, than always to find an organic explanation for paroxysmal epileptic fits. Both conditions rarely kill, and we only get an opportunity of a post-mortem examination in patients who have died of something else.

This way of looking at Raynaud's disease also gives us some clue to its frequent occurrence in women about twenty years old, for the nervous system is particularly unstable in women of this age, and it also helps us to understand why the disease is often met with in hysterical subjects. But wide as this view is it still leaves us in the dark as to why the condition is symmetrical and why it is paroxysmal.

Treatment. In the first place, and above everything, keep your patient warm, especially never let the fingers get cold. She must wear warm gloves indoors and out, and use a muff. She must wash as little as possible, and never in cold water. Should she suffer much, she had better always live abroad in some warm place in the winter. Keep her in the best general health possible. Nitrite of Amyl was tried some few years ago without much success, but a case we had in Miriam Ward a few weeks ago, and another case I have seen, seem to show that Nitro-glycerine is occasionally beneficial. The patient in Miriam Ward always had her attacks worse in the early morning, so when she first woke we gave a tabella

of Nitro-glycerine, and gave her one at intervals of an hour for three hours: under this treatment she decidedly improved, but I must add that I have seen it fail. It has been advised to put the hands in tepid water, to place one pole of a mild galvanic current in the water, and the other on some indifferent part of the body. We did this some years ago for a patient, and it certainly relieved the attacks at the time, but it did nothing towards permanently preventing them. Quite recently I have found Methylacetanilide, three grains every two hours given for six hours during an attack, decidedly beneficial in relieving the pain.

Prognosis. As is the case with some other diseases, although you will probably be very disappointed in your attempts to benefit the patient, and the case will drag on without any improvement till you feel desperate, yet in most cases, if the patient will carefully avoid cold—the great exciting cause—she will probably, in the course of several years, get less and less liable to the attacks, for it is not common to meet with the disease in old women. I wrote to a patient of mine who, while in the hospital, did not improve at all; and her father wrote to say she had gradually got quite well. If the attacks are severe, and you fear gangrene, you must be very strict in insisting on warmth.

Disinfectant Mouth Wash.—The following combination has been found both a pleasant and an efficient disinfectant:—

R.	Thymol...	gr.ijj
	Benzoic Acid	gr.xl
	Tincture of Eucalyptus			ʒijj
	Essence of Peppermint			℥x
	Alcohol...	ʒijj

- M. Sig.: Pour enough in a glass of water to render it turbid, and use as a mouth-wash.

Fermentative Dyspepsia.—

R.	Olei Creosoti	℥xij
	Spts. Tenuioris	ʒss
	Ammon. Benzoat	ʒij
	Glycerin.	ʒvj
	Infus. Caryophyl. q. s.			ad ʒvj

- M. Sig.: Tablespoonful in a glass of water.
(Dr. B. W. RICHARDSON.—*Med. Rec.*)

A CLINICAL LECTURE ON SOME CASES OF INJURY TO THE SPINAL CORD.

Delivered at St. George's Hospital Medical School,
Feb. 13, 1894,

By **WILLIAM H. BENNETT, F.R.C.S.,**
Surgeon to the Hospital.

GENTLEMEN,—In my last lecture* you may recollect that I did what I could to impress upon you the importance of regarding every injury to the head as a serious one if there were any signs of brain injury at all, although these symptoms may have amounted merely to a transitory giddiness or a slight dazing. I tried to impress upon you the fact, too, that insensibility is not a necessary consequence of concussion of the brain—that concussion of the brain may be associated with every degree of interference with brain function, from a mere momentary giddiness up to the profoundest unconsciousness. Recently, I happen to have had in the hospital a patient suffering from an injury to the spine which illustrates, in exactly the same way—that serious injury inside the vertebral column is not necessarily associated, in the first instance, with any gross evidence of injury to the spinal cord—a very important matter.

The patient I refer to was one H. C., a coal porter, who was admitted into the Oxford Ward on January 15th. He had been taking a sack of coals down an area, and as he was passing down the steps he slipped and fell seven or eight feet into the basement. For a second or two he felt that he could not move his limbs; but this loss of power, if it existed, was quite momentary. He got up, was able to walk up the steps with the help of his mate, and he was brought directly to the hospital. He walked up the hospital steps into the surgery, and was there seen by the house-surgeon, who was in doubt, at first, as to whether or not it was worth while to admit him. But, fortunately, as the man looked rather ill, he was admitted and sent to bed. I saw him two or three hours after his admission. He was then lying flat on his back, looking comfortable, complaining of no pain excepting around a tender spot about the root of his neck at the back. Upon placing

the finger where the cervical spine joined the dorsal vertebræ, there was certainly a tender area, but no swelling, no deformity, no gross symptom of any kind. *He complained as I was seeing him that he could not pass his water.* Now, although the man at that time *apparently* had nothing whatever seriously the matter with him, I was perfectly sure, for a reason that I will mention presently, that the very fact that he felt this inability to pass his water was quite sufficient, as I stated at the time, to show that he had a serious condition of affairs inside his vertebral column.

The next day when I saw him he had begun to lose power in his legs. He could move his legs perfectly well when I first saw him, and all sensation was perfect; but on the following day he was beginning to lose power, and another little symptom occurred—he had some feeling of “pins and needles,” as he called it, in both his upper extremities. Well, these symptoms progressed—loss of power in the limbs increased, and was coincident with loss of sensation. This loss of sensation gradually crept up his belly and finally reached a point, in 48 hours, about the level of his nipple on each side, being about an inch lower on the left side than it was on the right. So the case was clear enough now—that there was very grave injury to his spinal cord. At this time (as I thought I had diagnosed the nature of the injury, namely, that he had hæmorrhage into his vertebral column, which was exercising pressure upon his cord) I proposed to him that I should perform an operation in order to relieve him of the pressure produced by this blood-clot. Unfortunately, he could not himself make up his mind to let me have my way, and he wished to consult his wife first. She lived at a distance; it therefore took a long time for her to get here, and by the time she reached the hospital he was very much worse. The symptoms of pressure on the cord in the neighbourhood of the lower cervical and upper dorsal region were more pronounced, and for another day the symptoms continued to increase. He became still worse, and yet he would not allow me to perform an operation. The day after that—which brings us to the fifth day from the time of his admission—he had made up his mind that I should do as I pleased in the matter; but, unfortunately, at this particular time he *seemed* to get a little better. That is to say, he began to recover some sensation over the upper part of his belly, and gradually recovered all the sensation,

* “Clinical Journal,” April 4, 1894.

with the exception of a few spots, so far down as the anterior superior spines of the ilia. I say that was an *unfortunate* thing, because feeling that he was, *as he supposed*, getting better, he declined the operation once more. I pointed out to him at the time that it was an unwise thing, because I knew that this was the latest time at which the operation could be performed with any prospect of success. Twelve hours later the improvement ceased, and he began to get rapidly worse again. Sensation diminished, as it did before, by degrees the motor arrangements of his thorax became involved, and finally, a couple of days afterwards, he died. That is the case, and it is a very interesting one from several points of view. Here was a man who apparently when he came to the hospital, had little or nothing the matter with him. He was shaken, naturally he would be shaken, because he had fallen down seven or eight steps with a sack of coal on his back. But he had no signs of any serious spinal lesion at all then. The result which followed was hæmorrhage into the vertebral canal, between the dura mater and the bones; the blood pressed upon the cord, just as a clot of blood will press upon the brain, inside the skull, outside the dura mater.

This hæmorrhage, outside the dura mater, was sufficient, in the first instance, to account for the symptoms from which he suffered. And if he had been wise enough to have let me perform the operation I wished to do, he would have had a real chance of life, because, without any interference with his spinal cord at all, I could have got at this blood-clot, cleared it away, and relieved him of the pressure. This clot of blood followed the natural course that these hæmorrhages generally do. The hæmorrhage ceased, as was shown by the slight improvement which I mentioned, and the improvement, therefore, simply indicated that the clot was beginning to contract, as blood-clots always do when the bleeding ceases, and so he was getting some relief from the immediate pressure upon his cord. But I knew that this improvement was almost certain in his case to be deceptive and transient. Now, what was the cause of the relapse? because there was no further bleeding, the clot behaved properly, and at the post-mortem examination we had the opportunity of seeing that it was not very large,—although large enough to produce the effect in the first instance,—and showed no in-

dication of having been added to by any second attack of hæmorrhage. Well, what occurred was this—and this is the interesting point about this part of the case. The injury done to the cord by the pressure of this blood brought about myelitis—acute myelitis—which caused rapid softening of the cord, and accounted for these progressive symptoms of paralysis in the second instance, and that is why, in cases of this sort, when you are quite sure you have to deal with hæmorrhage,—as we were here—there can be no doubt whatever as to what the treatment ought to be. In deciding upon the treatment under circumstances like these, you should decide upon precisely the same grounds as those which guide you in the treatment of hæmorrhage in the skull, producing pressure upon the brain. Supposing a man comes into the hospital with an injury to the skull, being conscious on admission, and shortly afterwards becomes paralysed, perhaps, on one side of his body, with or without complete loss of consciousness, you naturally conclude that the man has compression from hæmorrhage, and there is no question about the proper treatment. The patient is immediately trephined, without the least hesitation. In the same way, supposing you have to deal with an injury to the spinal cord, such as existed here, you should, upon the same grounds, suspect the existence of hæmorrhage and treat it in the same way—the only way that it can be treated—by trephining, or, at all events, by opening the vertebral column and clearing out the blood-clot. So the cord can be relieved from pressure, and an immediate chance of life afforded, whilst the proneness to secondary myelitis is, at the same time, greatly diminished. There was no question, then, about the treatment in this case, but, unfortunately, the man would not submit to it, and all hope of life was lost. Whether I should have been really able to save his life or not is open to some question, but as to what ought to have been done there was no question at all.

In urging upon the patient the treatment that we wished to adopt, of course, there always is—there was in this case, and there is in all these cases—a certain difficulty. The operation of itself must necessarily be severe, and when you propose to perform a serious operation, the patient almost always wants to know—if he doesn't ask the question of you he is thinking of it—what the chances of saving life are. In other words, are

the chances sufficiently good to make it worth while to be subjected to an operation of such severity. Unfortunately, in these cases, you cannot promise very much *with certainty*. You can promise the patient that in the first instance you will relieve the pressure, but you cannot promise that secondary changes in the cord will not follow afterwards, although they will be less probable. That is the difficulty, and there is just that little difference between the case I mentioned just now—bleeding inside the skull—and a case like this. In the head case you can promise more certainly a good result, than you can in the spinal case, because vital secondary changes in the brain are not so liable to occur from slight pressure as they are in the case of the spinal cord. Your strongest point in urging the operation is, therefore, taken from you, because you cannot promise very much with certainty. So it happened in this case, that I could not urge the operation, perhaps, as strongly as I should have liked to do, because I did not feel justified in promising the man that I could surely save his life.

There are one or two other points in the case which are rather interesting. I said just now that when I first saw the man he complained that he could not pass his water. I also said that that symptom assured me he was the subject of some serious interference with his spinal cord. Now, that is a very important matter, because a knowledge of it sometimes enables you to diagnose a serious condition in a case, when there is really no other symptom whatever to guide you. Here is an instance—it occurred in my practice, and therefore I can vouch for it. A man was thrown from his horse whilst hunting, and, so far as he knew, suffered nothing at all. He was, of course, shaken, but he caught his horse, finished the day, and thought nothing more of the occurrence. Three days afterwards he came to see me—not on account of his back—he had nothing the matter with his back, so far as he knew—but because he could not pass his water. The natural inference was that the man had stricture, but on passing a catheter I found he had a perfectly healthy urethra. We then began to discuss the question of this fall a little more intimately, and when I heard what had happened to him—how, in falling, he had pitched flat upon his buttocks, felt a good deal shaken at the time, but nothing more, and that there then followed this inability to pass water—not all at once, but

by degrees, so that by the time he came to me he could not pass his water without pressing his hands upon the lower part of his belly, and in a sort of way squeezing it out, I was absolutely certain that the man in the fall had injured his spinal cord. You may take it as a truism which is well worth knowing, that it is absolutely certain when, after an injury to the back, no matter what other symptoms there may be, a patient cannot get his bladder to work—supposing, of course, that he has no stricture or any organic obstruction—that the spinal cord has been injured, and that it is your duty to insist upon the case being treated as a serious one. Just in the same way as I tried to show you it was your duty to insist upon a man who, after a fall upon his head, developed that peculiar pulse I mentioned to you, it was your absolute duty to make him lie up, so when this bladder difficulty shows itself in apparently trivial back injuries, it is your duty to insist on absolute rest, and to watch your patient most critically.

The patient I am referring to was at once ordered complete rest in bed. In a few days he began to lose a certain amount of power in his lower limbs, and he lost so much power ultimately that he could not move without help. Fortunately, the case had not, I suppose, progressed very far; he was wise enough to obey all the injunctions given to him, and in six months he was getting about again. When I last saw him, a year after the accident, he was walking fairly well, although there was occasionally a slight inclination to drag both his feet; it was quite evident, therefore, that he was still suffering to some extent from the damage to his spinal cord. Now supposing I had not recognized the importance of this bladder system, and had thought that the man, for some reason or another, had a little atony of the bladder, (I might very well have assumed that on some occasion he had been holding his water too long, and had, therefore, lost the power of emptying the bladder, as sometimes happens), I might simply have emptied his bladder for him from time to time, and might have allowed him to go about his work—might even, possibly, have let him hunt again. Had I done that he would have, undoubtedly, been dead by now. He would have developed a fatal myelitis to a certainty, instead of only getting this partial paraplegia from which he recovered. That case, then, is a sound illustration of a good practical point.

There is another symptom quite as important as the bladder symptom, though, of course, it is not quite so striking at first. In almost all injuries of this sort, where the damage is sufficient to lead to any ulterior change of a serious kind, you will find, either at the time of the accident, or, possibly, a few hours afterwards, that the patient suffers from certain "tinglings" in one or other of the extremities or about the trunk, generally the former. When, therefore, you are called upon to treat a man who has had a recent fall, inquiry should always be made very particularly about the sensations in the extremities. It is not a question of loss of sensation—that may come on later—but the question is whether the patient is conscious of any feeling of tingling, creeping, or "pins and needles." Next to the bladder symptom, these are the most absolutely certain signs of a dangerous condition of things. The peculiar sensations may pass off altogether—probably in many cases they *will* pass off—if you treat your patient properly, but if you neglect them you will be nearly certain to hear more of the case later on, supposing the injury is sufficient, because secondary changes in the cord will sooner or later occur.

Now for another important point. It does not follow that a severe injury is necessary to produce serious lesion of the spinal cord. It is curious how slight a blow or twist will cause grave symptoms. Take this case, for instance: A boy was playing cricket and received a very slight blow from the ball just above the small of his back, over the spine. He thought nothing of it at all—it did not disturb him in any way, and he went on playing to the end of the game. Nothing whatever happened to attract attention for six or seven days, but at the end of that time he began to lose power in his lower limbs. In ten days he had no power at all, and in a fortnight his case was given up as hopeless; he did not, however, die, but was permanently paralyzed below the waist. This was, undoubtedly, a case of myelitis, following upon very slight injury. Some years later he developed Potts' disease, for which I saw him. In describing the original accident he stated that he had a distinct recollection of a tingling sensation in his lower limbs a few hours after the blow. Had these tingling sensations received the attention they deserved the issue of the case might have been different—for the treatment would have been taken in hand earlier. So much, then, for that case in itself.

There was another case of a different kind altogether, because it illustrated a very severe form of injury to the spine, in the hospital under my care, a long time since—so far back as last July—of which I wish to speak. The patient was a man who, after a very severe crush, was brought into the hospital paralyzed throughout the lower part of his body—absolutely paralyzed. He had a fracture of the spine, the displacement being easily felt, and was very exaggerated. I did not see him at the moment of his admission—in fact, so far as I recollect, I did not see him until twenty-four hours afterwards. There was a large projection—the result of the fracture at the junction of the dorsal and lumbar regions. There was no question as to the severity of his case, and there came the question as to the treatment to be adopted. Ought we, in that man, to have attempted to reduce the displacement, or ought we not? That is always a very important question in the treatment of these cases of fractured spine. Well, in this case I did not reduce the displacement for these reasons: first of all, the man was so very bad at the moment of his admission to the hospital that it seemed probable that he had some other visceral injury, which would be sufficient to cause his death. (That impression afterwards proved to be wrong.) Secondly, so far as I could ascertain, by the absolute paralysis and the condition of reflex action and the other symptoms present, the cord itself was, undoubtedly, cut completely across by the displaced bone. If that were so, and there was really no doubt about it, it was clearly unnecessary to subject this man to the trouble of attempting to reduce this spine, unless it could very easily be effected. In order to have reduced it we must have given him some anæsthetic as he was in acute pain, and that would have been undesirable, because it would have thrown great stress on his lungs, which were already becoming engorged when I saw him; therefore, I rejected altogether any attempt at reducing this displacement, as it appeared to me a useless proceeding. Well, having declined to reduce the displacement on that account, it was clearly not justifiable to do what is recommended a good deal now, *i.e.*, to perform laminectomy—to remove the posterior wall of the vertebral canal, in order to relieve the spinal cord from the pressure of the displaced bones. I thought laminectomy unjustifiable, because, as I said before, I was certain this cord was completely divided, and, apart from this, the

displacement was so excessive, that by no ordinary operation of laminectomy—by which I mean the removal of the posterior wall of the vertebral canal—at the seat of the fracture could any good have been done. In order to have affected the purpose in view, we should have had to do more than the ordinary laminectomy, and this is one of my reasons for mentioning the case, because I want to show you what is proposed to be done in some of these cases, and what has been actually done. It is clear, even supposing the injury had not been sufficient, as, for instance, in a case like this (specimen shown), to completely sever the spinal cord, that the removal of the posterior arches of the vertebræ to any extent, would not have relieved the pressure, because you will see, the injury was really caused, and the pressure for the most part produced, by the sharp edge of the displaced anterior wall of the vertebral canal, at the point where the body of the vertebra is broken. In order to relieve the pressure in any of these cases, it would be necessary, not only to take away the posterior arches of the vertebræ, but to take away the angle caused by the projection backwards of the displaced bodies of the vertebræ. If this could be done, provided that the cord were sufficiently intact, all pressure could be relieved, and the patient could be given a chance of life he would not otherwise have. But you must bear in mind that this is a most severe operation—much more severe than the removal of any amount of the posterior wall of the canal would be, because the cord must be lifted from its bed and the projecting part gouged away. This operation would, therefore, be difficult and serious, so serious, indeed, that very few patients could stand it at all under the circumstances. In a case like that one I am mentioning, at all events, it would be quite out of the question. I wish you to understand that although you hear laminectomy talked of occasionally as a somewhat ordinary proceeding, and one which should, as a matter of course, be adopted, it is really useless in giving relief from the symptoms with which you have to deal in the vast majority of cases of fracture with displacement unless the removal of the projection formed by the backward dislocation of the bodies of the fractured vertebræ be effected at the same time. Propositions have been made in these cases, in which the cord is completely divided, to re-unite the spinal cord by means of sutures. That is not at the present time, at all events, an

operation which need be considered as practical; and whether it ever will be, I do not know. Now, in this case and in the other case which I have mentioned, there was a symptom which is of some interest, and which was common to the two cases. That was the symptom of priapism. Some of you do not seem to understand exactly what is generally meant by priapism, when mentioned in connection with injury to the spine. The priapism was most marked in the man who had the least severe injury—the man who had an injury about the lower part of his cervical region. He had very well marked priapism, by which, in an ordinary way, of course, you understand *erection* of the penis. You must, however, bear in mind that the priapism generally met with in these cases of injury to the spine does not, in the ordinary sense of the word at all, amount to erection. There is no *erection* of the penis at all. The organ is larger than usual, but flaccid, lying full and large on one side of the groin, is not hard, and certainly is not erect. The condition is simply a passive turgescence—the result of the removal of the inhibitory power—that is all it is. And this symptom existed in a marked way in both of these cases, which is, at first sight, a little peculiar, because you will find, if you refer to your books, that it is stated that this symptom, as a rule, is only met with in cases where the upper dorsal or cervical cord is injured—that it is rare in injuries about the lower dorsal region, and is seldom ever seen in injuries about the lumbar region. And that is generally true. But in one of these cases it existed, although the injury was between the twelfth dorsal and the first lumbar. Although, in a general way, this symptom of which we have been speaking is a passive symptom, simply a turgescence and not an actual erection, occasionally in these spinal cases an actual state of erection exists. Now, what is the reason of that? Why, generally in one set of cases should you get this passive engorgement, and why, in another case, should you get the active process? The reason is that, in the case of active erection irritation in the cord, actual irritation, exists, such as might possibly be produced at first by the irritation of a spike of bone. In the case of passive turgescence active irritation is wanting. One (the passive turgescence) is merely a symptom of interrupted conducting power, and the other symptom (the active erection) is an indication of actual irritation at the seat of injury.

So much has this irritation been at times that there have been a few cases—I have seen only one myself—in which, at the time of the passage of a catheter in order to relieve the bladder, there has been an involuntary evacuation of semen—quite unknown to the patient, not accompanied by any sensation, but a purely mechanical process, the outcome of an organ abnormally irritated, in the same conditions as it would be under natural circumstances of erection.

There was yet another point in this last case which, although quite usual, is worth your attention. The man was in the hospital six months, and in spite of the complete severance of the spinal cord, he remained well after he had recovered from his first collapse, almost the whole of the time, until within the last week. He ate well, got quite fat, and no bed-sore formed. Now, you know it is generally regarded as a distinctive characteristic of all these cases of injury to the spine that there should be a remarkable tendency to the formation of bed-sores. Here was a man with his spinal cord completely severed, and yet in spite of that no bed-sore of any sort formed, until about ten days before he died; and then all at once, without any apparent reason, a large sore on one buttock formed almost suddenly, and was followed by one on the other side. So great, indeed, was his tendency to bed-sores that he had a bed-sore on the anterior superior spine, where there was no pressure at all. Obviously, therefore, these sores which occur in some of these cases are not due to pressure in the ordinary sense of the word. It is not a question of mismanagement in the matter of nursing, or anything of that kind.

Now, coincident with the formation of these bed-sores, another thing happened. The man began to pass blood in his water—not that he had been improperly catheterized or anything of that sort—but his water became bloody, and soon pus came with it too, quite of a different kind to the mucous material he was passing before, and very shortly he began to get infiltration of urine into his perinæum, not because he had been torn by catheters, for he had simply had a soft instrument passed for the purpose of washing out his bladder. What is the explanation of all this? Why should these sores suddenly come, and why should this rapid change in the urine, etc., occur when the man all these months had been perfectly well? It simply meant this: that the secondary change which I mentioned to you just now as occurring in the first

case, and which is inevitable at some time or another in most of these cases, had commenced. And the secondary change in this instance took a descending direction, and very soon involved his lumbar enlargement, and with it his genito-urinary area. The moment that happened the functions of the kidney were disturbed, the tissues about his perinæum broke down, and the same process took place there as was seen about the buttocks and the anterior superior spine. From that moment he rapidly got worse and in less than a week he was dead.

These, then, are good indications to bear in mind when treating a case of that sort. If you find bed-sores rapidly forming without any ostensible reason, you may conclude that secondary changes in the spinal cord have commenced, and you may be then almost absolutely certain that within a very short time the implication of the kidneys will follow, and that the patient's end is not far off. As most of you know, perhaps, these changes in the urine, etc., were formerly supposed to be due to backward pressure upon the kidney in consequence of the inability of the patient to empty the bladder; an altogether mistaken notion. Indeed, even now it is not uncommonly found in post-mortem records that patients dying from fractured spine in the later stages have "surgical kidney"—an entirely erroneous description. The renal changes are due to interference with its innervation, and have nothing whatever to do with backward pressure from obstruction to flow of urine.

These cases which I have described, taken together, illustrate matters of importance which I should like you particularly to bear in mind. The point of greatest importance being the necessity of never treating lightly a case of injury to the back, no matter how trivial the cause may be. If there is the least indication of interruption of bladder power, or if there is ever so little tingling about the hands, feet, or trunk, such a case may prove most disastrous, and should be treated at once as being serious. There is frequently the same difficulty, as I pointed out to you the other day when speaking of apparently trivial cases of head injury, in getting patients to do exactly what you advise them to do. But, at all events, if you realize the possibly grave nature of the injury and advise them properly and they decline the advice, that is their affair and not yours. I am afraid, however, in some cases the fault may lie rather

the other way; the importance of these trivial symptoms in the first instance, not being always recognized by the practitioner, which must be my excuse for calling your attention to the subject this afternoon, in order that I may impress its importance upon your minds.

ORIGINAL ARTICLE.

ON THE TREATMENT OF FURUNCULOSIS OF THE EXTERNAL AUDITORY MEATUS.

By A. E. CUMBERBATCH, F.R.C.S.,

Aural Surgeon to St. Bartholomew's Hospital.

AMONG the minor affections of the ear, there is none which gives rise to such acute pain as furunculosis of the external auditory meatus, and the amount of misery and constitutional disturbance which it may cause must be seen to be believed.

The inflammation begins in a hair follicle, or gland, or in a group of these, and may be seated, either in the superficial, or in the deep layers of the cutis. It is chiefly confined to the outer portion of the canal, rich in gland tissue; but when severe, the inflammation may spread as far inwards as the membrana tympani. It often arises without any known cause, and may as frequently attack the robust as the weakly. No class is exempt from it.

Perhaps it most frequently occurs in connection with chronic suppuration of the middle ear, and chronic eczema of the meatus. It also often follows that dry and irritable condition of the meatus, which tempts a patient to use some hard substance, such as a toothpick, to relieve the itching. But any mechanical irritation of the meatus will produce furuncular inflammation, such as frequently syringing the ear, the instillation of astringent lotions, or the pressure of hard masses of wax, or other foreign bodies. It occurs oftener in adults than in children, and I have seen it more frequently in women than in men. It is most prevalent in the spring and autumn. Diabetic patients are said to be especially liable to this trouble, but I have not observed this to

be the case. The disease is said by Löwenberg to be due to the presence of micrococci, which find their way into the glandular follicles, and set up inflammation.

The intensity of the symptoms depends largely on whether the inflammation is situated in the deeper, or in the more superficial, layers of the cutis. Speaking broadly, the further removed the furuncles are from the external orifice of the meatus, the deeper seated they are, and the greater the pain and disturbance to which they give rise. But the intensest pain may exist when the boil is just inside the external orifice of the meatus.

The symptoms often begin with a feeling of fulness in the ear, and pain of a throbbing or tearing character, which gradually increases, often spreading over the side of the head and neck. The pain reaches an intensity at times almost unendurable, rendering it impossible for the patient to obtain rest without the aid of a narcotic, and sleep, even thus procured, is often broken and fitful. There is loss of appetite and feverishness. Movements of the jaw increase the pain, and the auricle is sensitive to the least touch. Tinnitus and deafness are only present, as a rule, when the meatus is narrowed or closed by a furuncle. When these symptoms are present without any obstruction of the meatus, it is in consequence of hyperæmia spreading to the tympanum and labyrinth.

When the furuncle is in the anterior wall of the meatus, the skin in front of the tragus may be brawny, swollen, and red, occasionally even livid in colour; when in the posterior wall, there may be swelling, redness, and tenderness over the mastoid, simulating inflammation of the mastoid process.

Early inspection of the meatus (and the speculum should be introduced with extreme gentleness) may reveal little amiss at first, and it may be only on touching with a probe that the skin at one particular spot in the meatus is found to be very sensitive. Soon, however, if the site of the furuncle is superficial, a sharply defined tumour is observed, and the skin over it is generally reddened. If, on the other hand, the inflammation is deep-seated, the swelling is ill-defined and only slightly, if at all, red. Often the furuncles are multiple, arising simultaneously, or at least rapidly following one another, and then they more or less completely close the meatus. In from two to eight days, the

inflammatory exudation breaks down, and an abscess forms. The more deep-seated the inflammation, the longer is the time required for the formation and evacuation of the abscess. Occasionally, though rarely, the inflammation subsides without the formation of matter.

After the abscess has burst, or has been opened, the pain quickly subsides, but often speedily returns, owing to the formation of another furuncle, or to some obstruction to the free escape of matter from one already formed.

On the subsidence of the swelling, there is frequently for some time a slight thickening of the affected part. Sometimes, in anæmic or cachectic subjects, and when the furuncles are multiple, flabby granulations spring from the abscess cavities, and as the discharge is more abundant, and the narrowing or closure of the meatus prevents a satisfactory examination, these granulations may be mistaken for polypoid growths springing from the walls of the meatus.

It is in such cases especially that there may be swelling and redness of the skin behind the ear, giving rise to the belief that the mastoid process is involved.

Such cases are always troublesome to treat, and make slow progress towards recovery.

There is generally a tendency in furunculosis to relapses at intervals, varying from a few weeks to as many months.

The *diagnosis* is not difficult. Hyperostoses, with the skin over them reddened and slightly discharging, may be mistaken for furuncles, but pain, if present, is slight, there is no constitutional disturbance, and touching with a probe will at once reveal that the tumour is bony.

Furunculosis is also distinguished from diffuse inflammation of the external auditory canal, by the uniform and concentric swelling, and the freer discharge, present in the latter affection. The proliferation and exfoliation of epithelium are also much greater in diffuse inflammation.

It is hardly possible to confound furunculosis with purulent catarrh of the middle ear; but it is well to remember that these diseases may co-exist, and that the latter may be the cause of the former, owing to the irritation produced by the purulent discharge.

The *prognosis* is favourable. The patient, who is probably much dejected and depressed by his suffering, and is apt to think, if the pain has been great, that the inflammation will spread to his brain,

or will produce permanent deafness, may be reassured on these points. It is well, however, to remind him that there may be a recurrence of the disease before complete recovery.

Treatment.—In the early stage of the disease, the primary object is to relieve pain, and shorten its duration as much as possible. This is by no means always easy. In most text-books the treatment recommended is an early incision, when the inflammation is deep-seated, under the belief that the pain is cut short by relieving the tension, and also that the evacuation of matter is thereby hastened. With this treatment I disagree. When the pain is not severe, an early incision is unnecessary; when it is severe, the skin is so exquisitely tender that, without an anæsthetic, it is hardly possible to make a free incision, except at considerable risk of wounding more of the ear than is intended, through involuntary movements of the head. Again, although an early incision frequently gives relief for a time, my experience is that, in the majority of cases, the edges of the cut quickly reunite, and the pain returns, often with increased violence. When matter has formed, in however small quantity, an incision undoubtedly cuts short the pain and hastens the cure, and care should be taken that the incision is a free one, and not a mere prick of the knife.

Next, avoid dropping Laudanum into the meatus. It stains the skin, thus interfering with a satisfactory inspection of the canal, and does not allay the pain.

Leeching also is of little use, except in cases of swelling in front of the tragus or over the mastoid.

Linseed meal poultices sometimes allay the pain, but often increase it. They are apt, too, to produce an oedematous condition of the pinna, further narrowing the orifice of the meatus, and Löwenberg believes they encourage the production of bacteria. On the whole, therefore, they had better be avoided.

The treatment I have myself found the most satisfactory, is, in the early stages of inflammation, to frequently instil into the meatus a Boracic Acid solution in equal parts of Rectified Spirit and water, gradually diminishing the water, till the patient can bear the application of the undiluted Spirits of Wine; to apply hot fomentations and Boracic poultices; and to gently insert elongated plugs of absorbent wool, soaked in Glycerine, to which a 20 per cent. Solution of Cocaine has been added. Where there is much swelling of

the meatus, before the formation of matter, these cotton wool plugs afford great relief, by preventing the contact of the tender and swollen surfaces.

As soon as there is the slightest indication of the formation of matter, a free incision should be made.

Should granulations arise after multiple or recurrent furuncles, they must be destroyed with a saturated solution of Chromic Acid or Perchloride of Iron, or by means of the galvanic cautery. The ear should then be syringed, night and morning, with warm Boracic Acid lotion, to which about one-third part of Rectified Spirits has been added, (the quantity, however, varying according to the sensitiveness of the meatus), and after gently drying, a plug of absorbent wool, thickly dusted over with powdered Boracic Acid should be inserted.

After all inflammation has subsided, the meatus generally remains dry for a time, with a tendency to the increased production of epithelial scales. To relieve this dry and irritable condition, the meatus should be anointed with Glycerine of Borax, or the Red Oxide of Mercury ointment, occasionally syringing with a weak antiseptic lotion, should there be any accumulation of loose epithelium.

Where there is a tendency to frequent recurrence of furunculosis, painting the meatus with a 10 grain solution of Nitrate of Silver will sometimes stop this tendency.

It is needless to add that constitutional treatment must not be neglected. If the cause of the attack can be discovered, it should, if possible, be removed. Errors of, or indiscretions in, diet must be corrected, and tonics administered where required. Arsenic and Sulphide of Calcium are recommended where no definite cause for the disease can be discovered.

THERAPEUTICAL NOTE.

Ichthyol in Nephritis.—Dr. Robert Coltman, Junr., of An Ting Hospital, Peking, China, writes as follows:

"Ichthyol has proved in my hands an invaluable agent in the treatment of three cases of nephritis, the details of which are here given:—

"Case 1. Man, æt. 25, entered hospital last April with general dropsy; eyelids swollen so

that patient could scarcely open them; limbs swollen enormously; large collection of fluid in the abdomen. Urine became solid on boiling in a test-tube. Gave 3 i (4 grammes) of Compound Jalap-Powder morning and evening for three days, then 3 Compound Cathartic Pills three times daily for three days more. This produced free catharsis, and the limbs and face became more sightly. He was then placed on Compound Scillæ Pills, two, three times daily, with an occasional dose of Compound Jalap-Powder. In May the abdomen was tapped with an ordinary trocar, and 400 oz. (12 litres) of clear fluid drawn off. Between May and September he was tapped again on three different occasions. On September 23rd, his abdomen being again distended, limbs swollen, and general condition bad, I decided to try Ichthyol as a last resort, without having any faith in it. I gave him one drop in water three times daily. Almost immediately the flow of urine markedly increased, the albumen diminished, the dropsy rapidly disappeared, and on October 10th he was dismissed apparently well, although the urine still showed traces of albumen. As little more than a month has elapsed, at the time of this report, I cannot say how permanent the improvement will be.

"Case 2. Man, æt. 45, shop-keeper. Has been treated as a dispensary patient for over a year for nephritis, with dropsy of the legs. On October 15th I began to give him Ichthyol, in drop doses, three times daily. On October 25th he was entirely free from dropsy, and the urine, which before was markedly albuminous, scarcely showed a trace. He is still on Ichthyol, the dose being reduced to $\frac{1}{2}$ drop twice daily.

"Case 3. Young man, æt. 18. Presented himself on October 28th, with dropsy of abdomen, legs, and scrotum. Urine markedly albuminous. Gave Ichthyol, $\frac{1}{2}$ drop, largely diluted with water, three times daily. November 4th, dropsy entirely gone, urine free from albumen, general condition much improved. Continued Ichthyol, in $\frac{1}{2}$ drop doses, to be taken for ten days longer."

(*Universal Medical Journal*.)

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WEDNESDAY, APRIL 18, 1894.

A CLINICAL DEMONSTRATION

ON A CASE OF

OBSTRUCTIVE JAUNDICE FROM MALIGNANT DISEASE.

Given at King's College Hospital, March 13, 1894,

By J. BURNEY YEO, M.D., F.R.O.P.,

Physician to King's College Hospital, and Professor of
Clinical Therapeutics in King's College.

GENTLEMEN,—This is a case of jaundice, as you can see, for "jaundice" simply means the staining of the skin and conjunctivæ yellow, from the presence of bile-pigment in the blood. The word jaundice simply means that the patient is thus rendered yellow. Jaundice is, therefore, a symptom rather than a disease, and we shall presently see that, in this case, it is a symptom of a very serious disease. But it has been said of jaundice, and with truth, that it is a symptom with symptoms. For instance, intense itching of the skin is a symptom of jaundice, this patient complains of it; great depression of spirits is another symptom, and often there is a considerable retardation of the pulse. In this case the pulse is not very slow, it keeps about 70. Loss of appetite is a common symptom in jaundice. All these symptoms doubtless depend on the circulation of biliary excrementitious substances in the blood.

Now with regard to this case, let me first tell you the few facts belonging to its history. The patient is a woman of 55 years of age, who has been occupied as a caretaker. She was admitted into the Todd Ward on February 19th, complaining of jaundice, emaciation, loss of appetite and strength, and aching pain in the epigastrium, especially after taking solid food. There is nothing in her past personal or family history bearing on her present illness, and I need not, therefore, trouble you with negative details. She has had, I may mention, a large family—ten children and two miscarriages.

Her present illness began last Christmas, with "gnawing" pain in the epigastrium. This pain became more and more severe and constant, so that she suffered, as she says, "day and night." Taking solid food aggravated the pain, and also

gave rise to great and distressing flatulent distension. She, therefore, avoided taking anything but fluids, and she grew steadily thinner and weaker, and on the 3rd of last month (February) she felt so weak, that she took to her bed. It was then that she first noticed that her skin was getting yellow. So you will please notice that the chief symptoms she complains of had preceded the appearance of jaundice by five or six weeks.

She has since lost flesh rapidly, and the pain in the epigastrium continues, and has extended round to the back. She has never, however, had any paroxysmal attacks—nothing resembling attacks of biliary colic. This is our patient's history—in the next place let us examine her.

In the first place, you see that her skin and conjunctivæ are intensely yellow. When first admitted she was of a bright lemon colour, now she is certainly much darker. Her face is thin, and its expression is worn and anxious, and her body generally is much emaciated. Her tongue is slightly furred, her pulse normal, about 70.

Let us at once examine the abdominal region, and here let me say that your physical examination of a patient should always begin with that part of the body to which his symptoms call special attention. Examine the other organs subsequently; but when a patient's symptoms point to the probable existence of abdominal disease, do not begin by examining the heart and lungs. I was once present at a clinical examination which was superintended by Sir William Jenner, and after the expiry of the usual quarter of an hour allowed to each case, one of the candidates asked Sir William Jenner for "more time," when Sir William Jenner replied that "if a man had not made out what was the matter with a patient in a quarter of an hour he never would!" A want of directness in your method of examining a case may, no doubt, lead to a great and unnecessary waste of time. We will not then concern ourselves at present about our patient's heart or lungs, or her reflexes, etc., etc., but go directly to the examination of the abdomen.

And first let us notice if mere *inspection* will teach us anything—well, not very much. The abdominal walls are thin and flaccid, and the coils of intestine are visible through them. There is no

visible abdominal distension, no dilated veins on the surface—the result of inspection is rather negative than positive.

Next, what can we learn by palpation? And here let me caution you how deceptive and delusive palpation of the abdominal region is apt to be. This you will readily understand when you reflect that in the abdominal cavity there lie coiled up 20 feet of small intestine, and 5 feet of large intestine; 25 feet of hollow tubing, sometimes full, sometimes nearly empty, or containing variable amounts of solid, liquid, and gaseous matters. These coils of intestine are more or less movable, and are frequently shifting their relation to adjacent parts. Then we have the stomach in the upper region, sometimes empty, sometimes greatly distended. The abdominal cavity, moreover, may be invaded from below by a distended bladder, or an enlarged uterus, or an ovarian cyst. It contains the liver and spleen, two organs which undergo certain changes of size, even in states of health; not to mention the gall-bladder. At the back of the cavity there lie the pancreas and kidneys. There are numerous glands in the mesentery which may become enlarged. There is a serous membrane, the peritoneum, which may become the seat of effusion, inflammatory or dropsical. Then there lies between your hand and all those organs a more or less thick layer of fat and muscle. When you remember all this, you will realize why it is that the exploration of the abdominal cavity has been regarded as proverbially difficult, and errors of diagnosis in connection therewith are common.

To avoid such errors as far as possible, it is desirable always to combine percussion with palpation, as I shall now do, and always to use extreme caution in making a diagnosis of abdominal diseases.

Palpation and percussion combined inform us that the liver is somewhat enlarged, its upper limit of dulness is somewhat higher than normal, and its lower edge can be felt an inch and a half below the costal margin. The edge is sharp, not rounded, and the liver substance does not feel hard or resisting, its surface is fairly smooth, but there is great tenderness over the whole of the right hypochondriac region and the adjacent part of the epigastric region. Occasionally some lumps can be felt in the right lumbar region, but these disappear after a purge, and they are probably faecal masses lying in a dilated colon. Percussion over the spleen, as you see, shows it to be distinctly

enlarged, and its right limit of dulness runs into that of the left lobe of the liver. Percussion over the stomach also shows some dilatation of that organ, and that it descends lower into the abdominal cavity than is normal. There is a slight amount of dulness on percussion, in each flank, from which we may infer that there is probably a small amount of dropsical fluid in the peritoneum.

This is about all the positive information we can get from an examination of the abdomen in this case.

Let us next examine the evacuations. Here is the urine, it is almost black from the presence of the colouring matter of the bile, and it gives, as you see, the characteristic play of colours when Nitric Acid is brought in contact with it as on this white plate. I will now boil some of the urine, and you see we get a slight opacity due to the presence of a small quantity of albumen: this is doubtless due to irritation of the kidneys by the passage through them of all this biliary excrement. I have also had the evacuation from the bowels saved that you may see it—you see that it is quite white—there is an entire absence of any colouring matter, and we may conclude that no bile whatever passes into the intestine. Here then we have evidence of complete obstruction of the bile ducts, and there exists *obstructive jaundice*. But we have next to consider how this obstruction has been brought about? I believe it is due to external pressure on the hepatic or common duct by a malignant growth.

It is clearly not simply *catarrhal*; its persistent and growing intensity, its long duration and the grave wasting cachexia accompanying it sets this quite on one side. It is not due to *gall-stones*; the absence of paroxysmal attacks, the great and increasing cachexia, the rapidly progressive loss of strength, the diffused tenderness and enlargement of the liver, and the onset of the illness with strictly gastric symptoms, viz., epigastric aching pain, pain after solid food, and flatulent distension, all point in another direction, and that is to the presence of a carcinomatous growth or growths in the vicinity of the hepatic or common duct.

The drawings I show you indicate the relations of the bile ducts to surrounding parts; you will see that a cancerous growth at the pyloric end of the stomach might readily come to compress the bile ducts; so also cancer of the head of the pancreas might compress the common duct; or secondary cancerous infiltration of the lymphatic glands in

the transverse fissure of the liver might cause compression of the bile ducts.

The symptoms, course, and physical signs in this case seem to point to the pyloric end of the stomach as probably the original seat of the malignant disease, and that it has infected secondarily the glands in its neighbourhood and the liver itself. The trunk and main tributaries of the portal vein are also in close proximity to the hepatic and common bile ducts, and we may expect soon to have further evidences of pressure on the portal vein in the form of ascites and possibly dilated superficial veins. There already seems to be a small amount of dropsical fluid in the peritoneum, and there is distinct evidence of splenic enlargement.

I am disposed to think that there may be also in this case some cancerous deposit in the hepatic bend of the colon, as the patient makes distinct complaint of pain in this region just before the bowels act.

We can do little in the way of treatment for a case of this kind, we keep the bowels relieved by non-irritating purgatives, and we allay gastric irritability by antacids and sedatives, and we give only such fluid food as can be taken without discomfort. You will, I hope, watch the progress of this case with interest.

A LECTURE

ON

TUBERCULOSIS IN CHILDREN.

Delivered in connection with the London Post-Graduate Course in the Hospital for Sick Children, Great Ormond Street, Feb. 22, 1894,

By F. G. PENROSE, M.D.

GENTLEMEN,—The point of view from which, in the case of children as in that of adults, we study the onset of tubercle has been entirely changed by the discovery of the *bacillus* by Koch. It has so altered our views that it is my intention to assume that *all* manifestations of tubercle are due to the growth and life-history of the tubercle bacillus. This is a view which, every year, is gaining in importance, though there are one or two men who keep an open mind, and are not willing to go so far as the more modern pathologists. Still, as a working hypothesis it is certainly doing much good, and helping us to follow

out the lines on which I think tuberculosis should be investigated, viz., that it is due to the growth and life-history of the tubercle bacillus within the body, together with the reactions of the body to that organism.

This leads us to consider, in the first place, how the bacilli effect an entrance into the body,—in the foetus, in the infant, and in the child respectively, because there are certainly differences in the different phases which we have to consider.

(1) Does the *fetus* ever get the bacillus directly from either of its parents? That is an extremely difficult question to answer. We have no direct evidence of the bacillus being derived from the paternal side; and in the human being there is very little on the maternal. Hensch, for instance, quotes a case of a child which died when it was 23 days old, with a large tubercular nodule in the brain. That is not an absolutely certain case, however, because it may have been infected after birth, and the disease developed with excessive rapidity. It is, at any rate, at least probable that it was a pre-natal condition, the bacilli which caused the tubercular nodule being received whilst the infant was still in utero.

In animals, however, we have a certain amount of evidence. In various places on the continent there are large slaughter-houses, where the animals are carefully looked over, and I have been able to find three or four records. Dr. Unna relates a case of a foetal calf with tubercle in the liver and lungs, which had been removed from the body of a cow with well-marked tuberculosis. Amongst the animals at the Institut Pasteur in 1889, Malvoz and Brouvier quote a similar case, where it occurred in the liver and lymphatic glands of an eight months foetal calf. These cases clearly show that the tubercle bacilli or their spores must have reached the foetus from the maternal organism, being carried there by the blood-stream from the mother to the foetus. The only other way in which it may possibly have occurred is, that leucocytes, containing live bacilli or spores, may possibly have migrated from the maternal organism into the foetal tissues and there deposited the tubercle which then develops. It is also possible for them to have been originally derived from the father.

Numerous endeavours have been made to try and settle this question of foetal infection experimentally, large numbers of pregnant animals being infected and the foetuses examined for evidences

of tubercle; but however thickly infected the parent animals were, in no single case has success been obtained in the way of infecting the foetus. So far, the evidence is very slight; and we shall find hereafter, clinically, that it is in strict accordance with what we should have expected. It is possible, as we have seen from the condition in the case of cattle, that foetal tubercle may occur; but it is excessively rare, and we have no absolutely indubitable proofs, so far as I can find, that it has occurred in the human subject.

Here we must digress to consider the questions of the tubercular and scrofulous diatheses, which are necessarily started during the growth of the foetus. Although these have not now the importance accorded them that they once had, we have, perhaps, of late years been rather forgetful of them; and it is well worth while to look back at the old classical account of Sir William Jenner's, published in the "Medical Times and Gazette" of 1860. When we consider the description of the two kinds of children we can understand why such a wide distinction used to be made between them, although our present inclination is to couple them both together as being children especially liable to be infected by the tubercle bacillus.

If we take the tubercular diathesis as Sir William Jenner described it, his description may be abstracted as follows: The nervous system is highly developed; the body is active; the adipose tissue is small in quantity; the organism generally is delicate; the skin is thin; the complexion is clear; the superficial veins are distinct; the blush is ready; the eyes are bright, with fine silky looking sclerotics; the pupils are large and the eyelashes long; the hair is silky-looking; the face is long; the child is good-looking; the ends of the long bones small, the shafts thin and rigid; and the limbs are straight. I show you here a child bearing out this description extremely well.

If you keep the appearance of this child in your mind for a moment, and then recall that of a scrofulous or strumous child, you will see what a difference there is. Sir William Jenner summarises the latter type as being of a phlegmatic temperament, with mind and body lethargic; figure heavy; complexion dull and pasty-looking; dirty-looking face; upper lip and alæ nasi thick; nostrils expanded; face plain and ugly; lymphatic glands perceptible; abdomen full; the ends of the long bones large, with thick shafts. Various pathological changes are present, such as strum us

ophthalmia, tinea tarsi, catarrhal inflammation of the mucous membranes of the nose, pharynx, bronchi, stomach, and intestines; and there is great liability to inflammation and suppuration of the lymphatic glands, together with obstinate skin diseases and caries of bone.

These are the two descriptions. They hold as good to-day as they did when first given; but, while fully conceding the differences, the modern conclusion is that the types have in common a diminished power to withstand the invasion or to prevent the spread of tubercle bacilli in the organism, or, in the terms of modern pathology, that the phagocytic and chemiotactic powers of the particular individual are weakened through heredity as regards its power of resisting the tubercle bacillus. In neither case is the bacillus actually present, and if only the individuals could be kept away from it, they would never become consumptive. Such a view as this was certainly not possible before Koch's discovery, which has not only enabled us to combine the hereditary failure of these two different types, but also to establish the essential unity of all the forms of tubercular manifestations in the different organs and tissues. A truth appreciated long ago by Laennec, but only established by Koch's discovery, in 1882.

Passing from the foetus to the infant, we have to consider in what ways the tubercle bacilli may gain entrance to its body. Of these, three are known certainly. They are (1) by direct contagion, (2) through the food, (3) through the air. There have been several cases, both in infancy and adult life, in which direct tubercular infection has occurred by contagion. The most striking of all occurring in childhood is one quoted, I think, by Mr. Eve, at a meeting of the Pathological Society, where a Jewish Rabbi in the East End of London, who was suffering from phthisis, inoculated several of the male children whom he circumcised, so that they got tubercular deposits in the lymphatics leading up from the penis into the abdominal cavity. This was so clear and definite a case that it may practically be accepted as proof. Traumatic cases in infancy may occur at any time. The eighth day after birth was the period in the instances just quoted; others may occur at other times, according to the chapter of accidents.

With regard to infection by food, the most important question in connection with children, is the infection through milk. In favour of

this we have a good deal of evidence drawn both from this country and from abroad. It is not absolutely conclusive; but it is extremely suggestive. For instance, Gerlach got positive experimental infection results by feeding calves, pigs, rabbits, and sheep on milk from cows whose mammary glands were the seat of tubercular disease. In 1883, Bollinger inoculated such milk into animals, and produced thereby general tuberculosis. In 1884, at the Medical Congress at Copenhagen, Bang reported finding enormous numbers of tubercle in milk from cows whose mammary glands were affected, and the successful inoculation of pigs and rabbits therewith. Martin, in the "*Revue de Medicin*" of 1891, showed that milk from cows kept in Paris or its suburbs frequently contained tubercle bacilli, with which he successfully inoculated animals. This is of considerable importance to us here in London, where so much "nursery" or special "milk for invalids" is sold, obtained, as I understand, from cows kept in London. This is probably about the worst form of milk that could possibly be given to our London children; as the cows kept in town for this purpose are more likely to produce milk that will engender tuberculosis than those kept in the country.

The large number of children with tubercle in the intestines or in the mesenteric glands, is in favour of the disease being started occasionally along the intestinal tract rather than elsewhere; though even in children, and from the very earliest age at which we come to get deaths from tubercle, the preponderance, at any rate in London, is certainly in favour of the chief source of infection being through the air. The lungs and bronchial glands, that is to say, are the original seats of infection in the larger number of cases. The sputum from tubercular people gets dried and blown about, so that houses, furniture, and one thing and another get contaminated, and so become a source of danger. We have the very interesting negative evidence that people who do not live in houses, such as nomad tribes like the Kurds of the Russian Steppes, the Arabs in Africa, never become consumptive so long as they continue houseless, but directly they settle down in towns or fixed abodes they immediately become liable to it. Hirsch, in his "*Geographical Distribution of Diseases*," has some very interesting remarks on this subject.

The periods at which children chiefly become

infected come next under our consideration. Cases in early life where the infection has occurred before birth or during the earliest period of childhood are, as I have said, not very common. Tubercle takes a certain amount of time to manifest itself; and although it is not unknown to have deaths under three months from tubercle, it is rare. Deaths during the next three months, *i.e.*, up to six months, are not uncommon. From the seventh month up to the second or third year is the most dangerous time of all, judging from the records in our post-mortem books.

In London measles has a very direct relation to the onset of tubercle in children. Amongst the London poor many a child passes through its attack of measles, but never seems to make a good convalescence, but gradually wastes, and within a short time,—a year or so,—dies of tuberculosis. The next great rise in the number of cases is out of the period of childhood, namely, in young adults, when acute pulmonary phthisis especially appears; but as that is beyond the period with which we are dealing I shall not touch on it.

Having seen how the bacilli enter the body, let us now consider their distribution, and very roughly compare the distribution in children with that in adults. Having gained entrance into the body it has somehow or other to get into the circulation. First of all, they appear in some cases to be caught in the lymphoid tissue, where they are for a time checked. This is strictly in accordance with what Metschnikoff has been teaching us with reference to other parasitic organisms; and we have a great deal of clinical and pathological evidence to bear this out. Amongst other things we generally find the earliest tubercle along the alimentary canal in the lymphoid masses in the sub-mucous tissues, and only after these have aggregated into collections of some size do the regular typical tubercular ulcerations form in the intestine, over the position of these lymphoid masses, by subsequent erosion of the mucous membrane. The distribution of tubercular lesions along the alimentary tract is very interesting. Tubercle of the pharynx and tongue is fairly common. Some years ago, at this hospital, Dr. Abercrombie worked out this point, and showed a very considerable number of cases in which rough ulceration of the pharynx and also of the tongue was due to tubercle. In the œsophagus it is excessively rare. Apparently, the food does not

stay there long enough, and its tissues are too resistant and thick for the tubercle bacillus to get lodgement in it.

In the stomach, tubercle is almost unknown. One example occurred in this hospital, quoted by Dr. Barlow, in the "Transactions of the Pathological Society" in 1887, in a case of general tuberculosis in which there were one or two small tubercular ulcers in the stomach, and in our *post-mortem* records there are one or two others.

When we come to the small intestine, as we get further and further down, cases grow more common until we get to the lower end. They are also extremely common in the large intestine.

If the leucocytes are not powerful enough to destroy them, and the bacilli get the best of it, they get, either by the lymphatics or by ulceration, into the general circulation; and, in accordance with the way the bacilli take, is determined the form in which the disease shows itself, according, that is to say, to whether it gets by dribblets into the general circulation and is distributed in various directions, or to whether it goes by ulceration or by the conveyance of the bacilli by leucocytes into the neighbouring tissues in which it is localized. One case has been recorded of the ulceration of a tubercular mass into the thoracic duct, the bacilli being thus poured directly in great quantity directly into the circulation, and setting up general tuberculosis. We are able to divide cases fairly clearly into two classes—(1) General tuberculosis, and (2) the various forms of local manifestation, tubercle principally affecting the pleura, the lungs, the abdomen, the glands, bones, etc.

The actual course is often difficult to trace; but clinical evidence certainly tends to support the supposition that general tuberculosis is distributed by the blood-stream. I have already mentioned the case in which there occurred ulceration into the thoracic duct; and it is well known, particularly to surgeons who have much to do with children, that shortly after operations on some tubercular bone, or for some other tubercular disease, it is not uncommon for general tuberculosis to set in, so that in many surgeons' minds the matter is considered as one of cause and effect; the tubercular focus has been disturbed, and an open wound having been made, the organisms get absorbed from the focus into the general circulation, and a general tuberculosis is started.

With general tuberculosis may be included

tubercular meningitis for this reason, that they are really two species of the same disease that run indefinitely one into the other. In quite an exceptional number of cases of general tuberculosis the life is ended by tubercular meningitis. On the other hand, if the meninges keep free the disease runs an extremely definite course.

Both these conditions are very much more frequent in children than in adults, though neither of them are confined to them. Tubercular meningitis, supposing we get it by itself, is a disease with very prominent symptoms and very rapid course,—three to six weeks; so obtrusive, indeed, are the symptoms, that once the disease has fairly set in they lead to a general masking of everything else; but one has to remember, at the same time, that tubercular meningitis is very seldom found alone. It is very rare to get it as a local disease without widespread tubercle in other parts of the body. It does so occur; there have been several cases recorded; but they are by no means common. On the other hand, it is excessively common for the life of a patient with general tuberculosis to be terminated by tubercular meningitis.

General tuberculosis is in childhood a very rapid disease, being usually of a month or six weeks' duration. There are only, pathologically speaking, a few portions of the body that escape deposit. The muscles, the oesophagus, the stomach, and the pancreas, are pretty well the only parts of the body that escape altogether; and even these are not absolutely certain to be free. It is generally possible to make a shrewd guess, but one cannot do more than that, about the organ in which the disease first started; and it is a well-known fact that in nearly all cases one finds a focus of caseous matter somewhere in the body. Apparently, the most frequent of all the primary foci are caseous bronchial glands.

Creasote in Phthisis.—There seems to be a steadily growing opinion of the value of this drug in tubercular affections, and the latest improvement in its administration is by combining it with Carbonic Acid in the form of a carbonate, the trade name for the compound being Creosotal; it is said to combine all the therapeutical advantages of the simple Creasote, with the avoidance of all the disagreeable properties. The dose is from ten to thirty minims, which may be largely exceeded by custom.

A CLINICAL LECTURE
ON
A CASE OF VARICOCELE.

Delivered at the London Hospital

By **O. MANSELL-MOULLIN, F.R.C.S.,**

Surgeon to the Hospital.

GENTLEMEN.—The patient to whom I wish to ask your attention this afternoon was admitted into the hospital for the radical cure of a small acquired inguinal hernia on the left side. As he lies in bed the external abdominal ring is open and free. As soon as he coughs or strains the intestine just protrudes through it. When he stands up the intestine descends a little lower, and what was not apparent before, the veins that accompany the spermatic cord swell up to a considerable size. There is a varicocele as well.

There is no difficulty in distinguishing the two tumours. There is an impulse on coughing in both, and they both disappear when the patient lies down and the scrotum is raised. But one vanishes suddenly, and with a distinct slip; the other so quietly that the progress is scarcely perceptible. The pressure of a finger upon the external ring will keep back one; it has no effect of any kind upon the other. A varicocele becomes full and distended when the patient stands up, no matter what pressure is used. The shape of the one is rounded and uniform; that of the other is irregular and characteristic; the veins can be felt coiling over each other as the cremaster contracts and raises them, much in the fashion that a bag of worms might.

The patient is only twenty-two years of age, which, I may remark in passing, is very young for an acquired inguinal hernia. He has never suffered from any trouble in connection with his varicocele; in fact he was not aware of its existence until a week or two ago. His testis is in normal condition; the scrotum is not much relaxed, and has no varicose veins upon it, such as you so frequently see in cases of varicocele. But he wishes to enter one of the services, and therefore has been advised to have the radical cure for both hernia and varicocele at the same time. The operation that I propose for this (so far as the repair of the muscular and aponeurotic wall of the abdomen is concerned) is that known as Halsted's, which is especially well-suited to cases of this kind.

The aponeurosis of the external oblique and the external abdominal ring are exposed; the various layers divided; the veins of the cord carefully isolated from the vas deferens, and excised, and then, while the vas is raised and kept well out of the way, the layers of the abdominal wall are drawn together in their proper relation to each other by six or eight deep mattress sutures. The cord, or rather the vas deferens, for this is nearly all that is left of it, passes straight out from the abdominal cavity, between the two outermost sutures, and lies upon the aponeurosis of the external oblique, covered only by the skin.

The cause of varicocele is not known. It is a congenital affection; people are born with it, though they rarely become aware of its existence until the generative organs begin to undergo their further sexual development at puberty. The reasons so often given for it, and especially for its occurrence upon the left side, belong to the class of what may be called surgical myths, venerable from their age, but for nothing else. The lower position of the left testis, the length of the spermatic veins upon the left side, the thinness of their walls (as a matter of fact they are extraordinarily thick, so that they do not collapse, but gape widely open when they are cut across), the left spermatic veins passing behind the sigmoid flexure or the rectum, and their opening into the renal vein with a more or less imperfect valve, while those on the right side open directly into the inferior vena cava, are interesting anatomical facts, but have nothing to do with the causation of varicocele. It is not the size of the veins only that is increased, or their tortuosity, it is their number, on which causes of this kind could have no effect.

The veins that accompany the spermatic cord are the remnant of the veins of the Wolffian body and duct in the fœtus. Their function disappears to a great extent, when the Wolffian body is superseded by the permanent kidney, and many of them disappear. Later in life, when the epididymis becomes functional, those that are left increase again. Those upon the left side of the body persist in larger numbers than those upon the right. Why they do so I cannot tell you, any more than I can tell you why the left umbilical and mesenteric veins persist, while the right disappear; or why the left thoracic duct is such a very much more important structure than the right ductus lymphaticus. When the viscera are transposed, and the arch of the aorta turns to the right, varicocele

appears on the right side, and not on the left; otherwise right varicocele by itself, or without a larger one upon the other side, is an anatomical curiosity.

Mr. Bennett has divided varicoceles into two classes: those in which the veins are few in number but of very large size, and in which the greater amount of swelling is high up, immediately outside the external abdominal ring; and, secondly, those in which the veins that surround the testis are especially affected; they are not nearly so large, but they are very much more numerous, and may almost conceal the body of the testis. A distinction of this kind cannot always be drawn, but in this particular instance the varicocele corresponds undoubtedly to the first of the two types, and as Mr. Bennett has pointed out the testis is normal.

I have, for a long time past, taken note of the condition of the testis in cases of varicocele, and I think there can be no doubt that in the majority the organ is softer and more flaccid upon the affected side than on the other, and in a very large proportion of cases smaller as well. In other words, the testis has not developed.

Varicocele does not cause atrophy of the testis, but it is associated with arrested development. If the varicocele attains any considerable (relative) size during childhood, the testis retains its infantile character. If this does not occur until puberty, the testis develops more or less according to the sexual age of the patient. If it does not make its appearance until the sexual organs are mature I do not think that it has any effect at all upon the condition of the testis. I have not been able, myself, to find out that it has any, and I cannot ascertain that others have done so.

It has been suggested that this arrest of development is due to the diversion from its natural course of the developmental energy of the tissues. This may be so, but it seems to me, on the whole, more reasonable to lay the blame upon the interference with the circulation. The blood in a varicocele is almost stagnant; the small stream that flows through the spermatic artery can have but little effect upon the immense column in the practically valveless spermatic veins, and, as a matter of fact, if these are divided the blood pours in a much larger volume from their upper than from their lower ends.

This is a point of some importance. If it is true, early operation, by improving the circulation,

should have some influence upon the growth of the testis. And I believe this is so. I have been able on several occasions to demonstrate a distinct improvement in the condition, if not in the size of the organ, after operation.

This patient had no symptoms. He was not even aware that he had a varicocele. In this there is nothing unusual. It is true of most people while they are in perfect health. Those, on the other hand, who have outgrown their strength, or who have been brought up in tropical countries: tall, thin men, with feeble muscles and a bad circulation, who become easily tired, and whose ankles are inclined to swell of an evening after standing; who get no fresh air, and take, and can take, but little exercise, may suffer very severely. And so may those who ordinarily enjoy perfect health, when they are run down and out of condition, especially if they are called upon for any prolonged effort. The scrotum becomes relaxed; the cremaster gives way; the testis hangs down, perhaps as much as six inches, below the crest of the pubes, and the veins are so distended that they contain many times their normal weight of blood. There is an aching pain in the loins, spreading down the thighs; the patient cannot hold himself upright; the testis is very tender, and occasionally there is severe local pain which has been described as due to spasmodic contraction of the tired-out cremaster. And often this is associated with a curious degree of mental depression and despondency, even when the patient has nothing whatever with which he can reproach himself, when he has never given way to disgusting habits or read viler literature.

Most cases of varicocele do not require any treatment. It is exceedingly common to be consulted about this condition by young men, but it is very rare to find a man of middle life who shows the least concern. When they do, it is often rather serious, for it frequently means that peculiar frame of mind which is associated with the development in late adult life, of what is called neuralgia of the testis, a complaint that is better severely left alone, and certainly is very rarely benefited, and still more rarely cured, by operation. But, putting these aside, and they ought to be kept in a class by themselves, a moderate degree of varicocele, occurring in a healthy, well-built adult, who leads a healthy life, does not require any special treatment. Cold sponging in the morning, or better still, night and

morning; avoiding constipation, which makes the varicocele worse, not because the rectum presses upon the veins, but because straining affects all veins that enter the abdominal cavity, and because constipation impairs health; wearing a light suspensory bandage if any very prolonged exertion is to be undertaken, but not as a matter of habit; and, in short, careful observance of ordinary hygienic principles will prevent its ever becoming a source of inconvenience.

The bandages that Mr. Bennett recommends, and in this I thoroughly agree with him, are of the simplest possible character. A piece of linen, sewn up into a kind of boat-shape, with tape coming from each end, and fastened round the waist, is as effectual and as comfortable as any. It costs next to nothing; it can be made easily at home; it is cool, and it is cleanly. As a temporary support a large silk handkerchief folded into a triangle answers perfectly. The apex of the triangle should be brought up in front, the other two angles behind; and the scrotum and penis should be held well up against the anterior abdominal wall. Appliances that will not wash and are costly for obvious reasons cannot be recommended.

But there is the other side to the picture. Varicocele may occur before or at puberty, and interfere with the development of the testis. It may, leaving older men and those who have lived much in the tropics on one side, cause a severe degree of pain, or prevent proper exercise being taken, or interfere with the patient's employment. It may grow so large as to be in continual danger of rupture or thrombosis; or it may, even when it is very slight (for in this there is often a curious amount of discrepancy), prevent a man entering one of the services. Under these circumstances there is no doubt a radical operation should be advised.

There is only one method now—the open one; all the older plans of acupressure, subcutaneous section, galvano-cautery, etc., are discarded. A sufficient length of the veins, with the fascia that surrounds them, is excised between two ligatures, and then the cut ends brought close together, so as to sling the testis up and allow the cremaster to recover.

All the ordinary steps to ensure thorough cleanliness are taken; the patient is anesthetized, and an incision an inch and a half in length is made from over the external abdominal ring downwards,

almost to the scrotum. The inter-columnar fascia is divided, and the veins with some scattered cremaster bundles at once protrude. The vas, which lies behind, is carefully isolated, and then a silk ligature passed around the whole group of veins, still enclosed in their sheath of fascia, immediately below the external abdominal ring. Then the veins can be gradually drawn out, separating the vas from behind them as they protrude, until a sufficient length has been isolated. The second ligature is placed around them; the intermediate portion of veins and fascia excised; and the ends of the ligatures above and below fastened together. Dusting a little Iodoform into the wound and closing it with a suture or two completes the operation. No drain is required, and as a rule no vessel requires ligature.

I prefer silk because there is less risk of its slipping, and in the case of a large varicocele with many thick-walled veins the amount included is often considerable. Transfixion, which would prevent this, is not advisable, as a vein might easily be pierced. Afterwards, when the patient is placed in bed, the scrotum is raised and an ice-bag applied for 24 hours. I have never known inflammation of the testis follow, but there is always a certain amount of tenderness and congestion which is relieved by this.

The wound heals within the week, but a little hard mass can be felt underneath it for some months, formed probably by the lymph that collects around the ligatures.

When the operation is performed in this way, the spermatic artery, as Mr. Bennett has shown, is probably always included and cut away. It does not run with the vas deferens, at any rate when there is a varicocele, but in the middle, among the veins. I have never known any ill result follow. The deferential artery probably has sufficient collateral communications in the epididymis to maintain the nutrition of the whole organ.

Solatica.—

R. Opii Pulv.
Ipecac. Pulv. ... aa gr. xij
Sodii Salicylat ... 3j
Ext. Cascaræ Fl. q. s.

M. Div. in pil. No. xij

Sig.: One or two pills for a dose.

(Dr. BENJAMIN WARD RICHARDSON.—*Med. Rec.*)

A CLINICAL LECTURE ON THE TREATMENT OF TRANSVERSE FRACTURE OF THE PATELLA.

By GEORGE COWELL, F.R.C.S.,
Senior Surgeon to the Westminster Hospital.

GENTLEMEN,—The treatment of transverse fracture of the patella has exercised the ingenuity of surgeons from very ancient times. The injury is a very common one. There is great difficulty in counteracting the separation of the fragments, which is caused by the contraction of the powerful quadriceps extensor, in the tendon of which the patella is placed. There is, also, the liability to infection and inflammation of the important joint, whose synovial membrane is nearly always torn in this injury. Whilst, therefore, the main object of the treatment is to keep the fragments in apposition in order that bony union may be obtained, there is always the risk to the joint to be considered and guarded against, lest the mechanical means we adopt light up mischief which may prove more serious to the patient than the non-osseous union of the fracture so often resulting under the ordinary treatment. The great multitude of methods suggested bears witness to the almost insuperable difficulties that exist in devising a plan which will combine the completeness of apposition with a minimum of risk to the joint. It is a question if it is worth while to run any risk in these cases, when one remembers the very useful limbs that result from ligamentous union. In fact, for a long time it would seem to have been the object of the surgeon in the hospital treatment of these cases to make use of such means as would result in obtaining the shortest possible ligamentous union, and with the least danger to the limb. This appears to have been best obtained by relaxing the anterior muscles of the thigh, by keeping the leg extended upon a splint, with or without elevation of the limb, and by the application of direct pressure upon the fragments of the fractured bone, at once or after the swelling in the knee has subsided. In carrying out these two objects there is room for much variety of detail. You have seen the simple plan adopted in this hospital in some of the cases, and old patients present themselves from time to time showing how useful limbs are with a short ligamentous band between the fragments. It is

generally wise for the patient to wear a knee-cap for twelve months or so as a support in these cases, though I admit that it is not easy to get working men to do so, as the apparatus handicaps them in their work. When it is not worn, it is found that unless the ligamentous material is of good width it gradually becomes elongated and attenuated, until I have seen the fragments six or seven inches apart, the upper fragment being so drawn up as no longer to be any defence to the knee-joint; and yet I have been surprised to find how useful the limb was under the circumstances, and how well the patient could walk upon it.

In consequence of this not infrequent result, there has long been a *desideratum* for some means for keeping the fragments in close apposition, at all events in the treatment of artisans. And this induced Malgaigne to devise his hooks. I remember seeing them tried in several cases, when I was a dresser, and subsequently in Paris. There were three great objections to them, and they have quite fallen into disuse.

1. They were exceedingly painful.
2. They set up considerable and even dangerous inflammation and ulceration.
3. They often failed to procure bony union, because they tilted the fragments and rarely kept even the edges in apposition.

Dieffenbach suggested another plan, viz., the division of the tendon of the quadriceps extensor close to the upper fragment of the patella. He then placed the limb on a splint, and applied strapping and bandages. This method was by no means always followed by osseous union, and for many reasons is now rarely adopted.

I may mention another plan which I think was adopted by Mr. Lund of Manchester, viz., the passing of a specially constructed steel hare-lip pin transversely through the centre of each fragment, carefully avoiding the synovial surfaces, and then fixing the fragments in apposition by a twisted suture round the pins on either side, a ham splint being fixed to the limb at the same time. This plan often succeeds if the pins are passed evenly through the fragments so as to lie in the same plane; otherwise some tilting of the fragments will occur. Unfortunately, there is a risk sometimes of an undue amount of inflammation being set up in the track of the wound, whereby the result may be marred.

The introduction of the antiseptic treatment of wounds gave a great impetus to the operative

mechanical treatment of patella fractures. The plan of laying open the fracture and the wiring together of the fragments had, it is true, been adopted by a few American surgeons prior to the systematic use of antiseptics; but there is no doubt that it was the use of antiseptics that first gave surgeons in this country confidence in this heroic method of treatment in other than compound fractures. It is adopted in two classes of cases, viz., in recent fractures, and in old cases where the ordinary treatment has resulted in a considerable separation of the fragments. A long longitudinal incision should be made, and the fragments exposed. All blood and serum should be squeezed out; the anterior margins of the fragments should be drilled, and the fragments firmly fixed together with silver, or better with iron wire. The wound should be carefully closed and dressed; the limb should be bandaged from the toes to the middle of the thigh, and a splint should be applied to the back of the limb. With careful antiseptic treatment the wound ought to close quickly, and the wires may become encysted and be allowed to remain. If suppuration does occur in the track of the sutures, it will probably be necessary to remove the wires in two months after the operation. This plan generally succeeds in bringing about osseous union; but except in compound fractures and in old cases where ordinary treatment has resulted in three or more inches of separation between the fragments, with inability to use the limb, it is a question whether a surgeon is justified, unless he is fully accustomed to the use of antiseptics and confident in his ability to apply them throughout the case, to convert by a surgical procedure an injury which may be treated with fair efficiency by simple means and without danger, into one which may easily become dangerous to the limb and even to life. I always say, Gentlemen, that this is a proceeding we can adopt almost with impunity and with satisfaction in the wards of a hospital, where we have constant supervision and every appliance at our hand; but one which would be quite unjustifiable amid the routine of ordinary practice, unless the surgeon is an expert in the use of antiseptics, with time and assistance to carry it out.

On the other hand, in compound fracture of the patella with displacement, this is the best treatment that you can adopt. The accident in itself is one of risk to life and limb, and you hardly increase that risk by your treatment, whilst the usefulness of the limb will depend upon its adoption.

Again, in old cases where a useless limb is the result of the ordinary treatment, a bold proceeding of this kind is indicated. A case in illustration of this has just occurred in Northumberland Ward. A strong heavy man in the prime of life had fractured both patellæ transversely some four months previously to his admission. He had been treated in a Cottage Hospital for a long time, probably in the ordinary way, with the result that he had been discharged with fair ligamentous union in one bone, the fragments being about an inch apart but firmly held together by a broadish band. In the other knee, however, there was a separation of about $3\frac{1}{2}$ inches or a little more, and apparently with no ligamentous union at all, the upper fragment appearing to be tilted with the fractured surface towards the joint and fixed by adhesions in that position. The limb was useless, as it would suddenly double up under the patient and bring him to the ground.

In this case the useless limb was subjected to the operation under discussion. A long longitudinal incision was made, and the two fragments dissected from their adhesions, and a thin irregular layer of bone was cut with a fine saw from both the fractured surfaces. Great difficulty was experienced in turning the upper fragment into its proper plane, and the stout silver wire that was first used was speedily fractured, and stronger iron wire had to be substituted. The fragments were, however, secured firmly in apposition. The man did well, and there was very little swelling and no rise of temperature. The wound healed quickly, but later on some suppuration occurred in the track of one of the two wire sutures that were applied, and it had to be removed; but the other still remains in the bone. There is firm osseous union, and the limb is a most useful one. There is still some stiffness of the knee, but it will probably gradually diminish. I have every reason to be satisfied with the result in this case, and I give it as an example of the class of case in which I would advise you to adopt that method of treatment.

With regard to the complete inversion of the upper fragment in this case, I think that it is instructive. It teaches us to moderate our methods of forcing the fragments together. Strapping is often used for that purpose and very usefully, but it must never be applied too energetically. We must rather aim at a gradual approximation of the fragments, and not a rapid and forcible one. To

tilt the fragments defeats the object that we have in view.

Amongst the many methods of suture or rather ligature that have been devised, there are two others that I should wish to describe to you. Mr. Barker's plan was described at length in the "British Medical Journal" of February 20th, 1892. Every antiseptic precaution must be taken. He says: "The lower fragment of the patella is steadied at either side by the left forefinger and thumb of the operator, who stands on the right side of the patient. A narrow-bladed knife is then thrust into the joint, edge upwards, exactly in the middle line of the ligamentum patellæ at its attachment to the lower fragment. As this knife is withdrawn cutting upon the lower edge of the patella, the skin wound is slightly enlarged to about two-thirds of an inch. Through the wound thus made a stout-handled pedical needle is thrust into the joint behind the lower fragment, and is pushed upwards behind the upper fragment, and through the quadriceps tendon in the middle line as close to the border of the bone as possible. The upper fragment at this moment should be forced down and steadied. When the point of the needle is seen under the skin, the latter is drawn upwards and an incision is made upon it and for two-thirds of an inch downwards to the edge of the patella, the knife entering the joint in the middle line over the needle. The eye of the needle is now threaded with stout perfectly sterilized silk or wire, and is withdrawn, carrying the thread of course behind both fragments. The needle, again unthreaded, is now passed through the same skin wound below and out of the upper wound, but this time in front of and close to both fragments. Here it is threaded with the other end of the suture, and is withdrawn. The thread now forms a loop over the upper border of the upper fragment, both ends hanging out of the lower wound, one arm of the loop passing, of course, behind, and the other in front of both fragments. The upper fragment is then forced down by an assistant until its broken surface touches that of its fellow, against which it is rubbed by lateral and antero-posterior movements, until it is felt that any blood clot or other material is dislodged, and they are both in position and grating. The operator then pulls firmly upon the suture, and ties or twists its two portions upon the lower border of the patella. The projecting ends are then cut off, and the small skin wound closes over the knot. Neither of the

wounds above or below the patella requires drainage or suture; they are simply covered by an antiseptic pad. The whole joint is then enveloped in Salicylic wool and evenly bandaged, and the limb is put up on a long back splint. When the wounds are made into the joint, as much blood and clot are squeezed out through them as possible."

In this method of treatment there is the same necessity for complete asepsis owing to the serious risk of acute mischief before mentioned. To my mind there is in the method this serious addition to the risk, that the suture passes through the joint. I have never tried this method because the risk of arthritis and a stiff joint seemed to me to be too great.

The other plan I have tried myself, and find it free from some of the risks of these methods of suture. It is described by Mr. Herbert Butcher in the "British Medical Journal" of April 30th, 1892. The ligature for holding the fragments together, instead of being placed longitudinally in the middle line and passing through the joint, encircles the margin of the fragments and is altogether outside the joint, the tendons above and below the fragments being transfixated from side to side instead of from before backwards. The procedure that I have adopted, and it should be done if possible within twelve hours of the fracture, is as follows:—

The operation should be performed with strict antiseptic precautions. An assistant holds the fragments in apposition, and a short incision of half an inch is made in the skin on each side of the patella, opposite the ends of the line of fracture. Through these wounds all blood and clot is squeezed out. A stout curved pedical needle on a handle is entered at one of the incisions, passed by the side of the upper fragment of the patella, through the tendon of the quadriceps extensor, and then, depressing its point, along the other side of the patella and through the wound on the other side. The needle must be kept as close to the bone as possible in its whole route. The needle is then to be threaded with stout sterilized silk and withdrawn, drawing the silk round the patella. The needle again unthreaded is then entered at the same wound as before, and passed round the lower fragment, piercing the tendon of the ligamentum patellæ close to the bone in the same way. The needle emerging at the other wound is threaded with the silk and

withdrawn. The ligature then surrounds both fragments of the patella, both ends of the ligature protruding from the same aperture in readiness for tying. The two fragments are then rubbed together to press out all blood and get perfect apposition, and the ligature is tightened and tied, the knot falling into the wound and lying by the side of the patella. The very small wounds may be treated each with one small horse-hair suture, and a pad of absorbent wool. The limb should be bandaged, and a ham splint applied, and the patient kept in bed. The dressing need not be touched, and as a rule neither pain nor discomfort occurs. I have tried this method in five cases. In four there was no rise of temperature, the wounds at once healing and firm osseous union and a moveable joint resulted. In one case there was some suppuration in the track of the ligature from, I suppose, some failure in the asepsis. Several superficial abscesses formed in the neighbourhood of the knee, and considerable swelling about the joint. The patient was a woman of between 40 and 50, and it was found impossible to get the sinus to heal. Osseous union took place, and ten weeks after the operation the sinus was scraped out and the ligature searched for without success. Perhaps not a very diligent search was made, as it was thought probable that the ligature had disappeared. This treatment did not succeed in healing the sinus, and some six weeks later an end of the knot of the ligature presented at the small discharging wound. It was seized by a pair of forceps, and the ligature was divided and removed, the silk being as sound as when it was introduced. The wound then rapidly healed, but the knee was a stiff one when the patient was discharged. This case was interesting as showing how long a time silk will remain unchanged in the presence of suppuration. In spite of this one less fortunate case, I can recommend this plan of treatment if you can thoroughly carry out the antiseptic method; if not, I should strongly advise you to be content with the ordinary treatment of bandage and splint.

Tabes Dorsalis.—For lightning pains, Phenacetin, 0.50 gramme ($7\frac{1}{2}$ grs.) in wafers every half-hour until 4 grammes ($3\frac{1}{2}$) have been given. If not well borne, Phenazone hypodermically, or Hydrochlorate of Morphine combined with Sulphate of Atropine hypodermically.—(GRASSET, *Journal de Médecine de Paris*, No. 48, 1893).

CLINICAL NOTES.

(Specially reported for *The Clinical Journal*. Revised by the Author.)

WITH MR. JONATHAN HUTCHINSON AT THE CLINICAL MUSEUM, APRIL 3, 1894.

THE following were the principal cases which attended for demonstration at Mr. Hutchinson's lecture on Tuesday, April 3:—

Lupus Vulgaris in numerous patches scattered over the Face and Limbs.

The subject of the case was a fine healthy-looking lad of 16. He had upwards of twenty separate patches of common lupus in the exfoliative condition. In addition to these he had many sound, supple, white scars which had been produced by treatment. The disease had begun eight years ago by a crop of what his mother described as "blind boils after measles." He was one of ten children, and his parents and all their offspring were in good health.

Remarks.—(1) When lupus vulgaris is multiple, you may be sure that the multiplicity was attained at the very onset. After it is once well established, excepting satellites, no new patches are produced. There is a short period of liability to general infection at the onset; and then the disease settles into a quiet course, the patches spreading at their edges, but no new ones being produced. (2) The subjects of lupus vulgaris are often in good health. There is no risk that it will infect the viscera with tuberculosis. (3) The subjects of lupus vulgaris are not unfrequently of healthy families. (4) The treatment should be by the destruction of the growing tissue. In the present the numerous healthy scars prove conclusively that local measures were sufficient, and all that was needed was perseverance.

A Case of Chancre of the Tonsil.

The patient, a lad of 20, admitted sexual exposure to risk, but he had never had any sore on his genitals, and his first ailment was an ulcer in the throat. He was now covered with a dusky papular rash. There were several large hard glands in the left side of his neck, and none on the right. The affected tonsil was much enlarged and somewhat indurated. Its fellow showed no sore.

Remarks.—The diagnosis of chancre of the tonsil is usually attended by some elements of doubt, since the sore may be a secondary one. If it be a chancre it ought to be on one side only, with a glanular bubo on one side only of the neck, and there ought to be a clear history of its having preceded by some weeks the eruption on the skin. The absence of a sore on the genitals goes for very little, since not unfrequently the chancre is never found.

Ichthyosis with Papillary Growths, and tendency to the production of Horns.
(Mr. J. Hutchinson, jun.)

The child, a boy of 2, showed very large patches of papillary ichthyosis, rough, spinous, and black. The peculiarity of the case was that the patches, although so large as to almost involve the whole surface, were yet abruptly margined. His face, chest, and abdomen were almost free, but the skin even here was harsh and dry. In the palms of the hands and on the borders of the ears were many little horns. Nothing was known as to the child's family history. The condition was, of course, congenital. Many portraits were shown illustrating various forms of ichthyosis, but none exactly like the child, since in the latter the lesions of the skin were symmetrical, yet not universal, and showed no tendency to the common arrangement in streaks. Both nipples were involved in papillary growths, and the scrotum and penis were covered. No definite deviation from bilateral symmetry could be discovered. Treatment by shaving was recommended, and it was particularly urged that no horns should be allowed to grow. In enforcement of the latter point, some drawings showing enormous developments, the result of neglect, were exhibited.

Raynaud's Malady with definite tendency to diffuse Scleroderma (Morphœa) of Face and Hands.

The history of this patient is recorded in "Archives," vol. iv., p. 177. An interval of more than two years had passed since the beginning of the malady, and eighteen months since the first notes. Mr. Hutchinson stated that he was indebted to Mr. Clarke, of Upper Clapton, for the opportunity of observing the patient. The case was one of very great interest, as a con-

necting link between morphœa and Raynaud's phenomena. There could be no doubt that the fingers, hands, and face were hide-bound. The face was stiff and drawn, and showed some stigmata, and the fingers were wooden and thin. These conditions were, however, less developed than in many cases. The ends of most of the digits had been lost by gangrene, and one forefinger was at present time in a state of sphacelus. The disease had set in rather suddenly in a previously healthy woman (æt. 32), and in consequence, apparently, of severe sorrow. Very great benefit had resulted from the continued use of small doses of Opium. The extreme susceptibility to changes of temperature, with great pain in the digits, had quite ceased, and the patient had regained her health. The scleriosis changes had, however, not ceased.

An Example of the recurring form of Spindle-celled Sarcoma of the Skin
(Dr. HOOPER MAY, of Tottenham).

The patient, a gardener, aged 36, had recognized the growth for nine years, and attributed it to a blow from a cricket ball. It was placed on the inner part of the left thigh, and consisted of a congeries of bossy, smooth lumps, some of which were slightly constricted at their base. As no operation had ever been performed, there was in this instance no proof of tendency to recurrence. Mr. Hutchinson, however, remarked that it was a kind of growth which always showed the most inveterate tendency to return, and he believed that the best practice was to let it alone. It would slowly spread, and probably in the end show malignant tendencies; but its growth would be much slower than if excised. Several cases illustrating these statements were narrated. In this instance, although the area involved was as large as an outspread hand, yet it caused the patient but little trouble. In reply to a question, it was stated that the lumps, although bossy and smooth, had nothing to do with keloid; and that, although the disease was often attributed to a bruise, it never developed in a scar. It was one of the most peculiar forms of primary sarcoma of the skin, and all the examples of it were alike.

Crateriform Malignant Growth on the Lower Eyelid (Mr. J. HUTCHINSON, jun.)

This case was that of an old man of 64. On his left lower eyelid a bossy lump, as big as the

half of a small walnut, had developed in six months. It was firm, but not very hard, and as yet had not ulcerated. It was loosely adherent to the periosteum. It was not in the least like the ordinary forms of rodent ulcer so common in this part, and, as was pointed out, it had grown far more quickly than they usually do. Although no crater had as yet formed, yet the growth was probably of that character. A number of illustrations of the crateriform ulcer were produced for comparison. Although this peculiar form of growth is undoubtedly epithelial cancer, yet it seems to have little or no tendency to return after excision, and, as a rule, does not implicate the lymphatic glands.

Short Broad Fingers in a Boy (Dr. Stocker, of Forest Gate).

This boy had very peculiar digits of both hands and feet. They were so broad as to much resemble those of acromegaly. But they were short also, and the condition was congenital. The hands were dusky, from feeble circulation. The boy said that his school nickname was "Froggy," and the appearance presented by his short thumbs was really very suggestive of a frog's foot.

Remarks.—It is important to distinguish cases like this from true acromegaly. Probably the condition is a matter of inheritance.

Brachioptegia (?) in an Infant, with Stiffness at the Shoulder (Dr. Stocker).

In this case the child had been delivered by a midwife, after a difficult labour. Although now three months old it had never used its left arm. It appeared to be very stiff at the shoulder joint, though not ankylosed. There did not appear to be the slightest pain. Although somewhat wasted there were no defects of nutrition or circulation in the limb, and the child could grasp with the fingers. The age of the child prevented any attempt to estimate sensation. It was certain that the muscles, although small, were not wholly atrophied. Mr. Hutchinson stated that the diagnosis lay between injury to the upper part of the humerus and brachioptegia from rupture of nerve-roots. He was rather inclined to the former. It was clear that some injury had occurred during delivery. The child was in perfect health.

WITH DR. J. EDWARD SQUIRE IN THE NORTH LONDON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST.

Anæmia and Phthisis.

HERE is a case of pulmonary phthisis with no very noteworthy features, either in the symptoms or course of the disease. Yet it is worthy of a few words of comment, because it is an example of a very common history.

The patient, a young woman in business, first came under my notice elsewhere as an out-patient. She complained of little that was definite, giving such an account of herself as we are accustomed to hear from the anæmic girls who attend in such numbers in any out-patient room. Like so many of these anæmic patients she had a slight cough, not prominently mentioned amongst her complaints. Examination of the chest revealed an unsuspected patch of tuberculous deposit in the left apex.

The mischief extended instead of resolving, and I, therefore, had her admitted here. The chest now shows diminished resonance at each apex, only above the clavicle on the right side, but extending much further down on the left. Over the whole upper lobe on the left side we hear dry crackling râles, and there are some creaking sounds in the right apex.

The mischief had involved the whole of the upper lobe of the left lung, and the apex of the lower lobe; the apex of the right lung was also affected. She has improved considerably since her admission here, and the disease seems now to be quiescent in the lung, but there is some commencing mischief in the larynx.

I draw your attention to this case to emphasize the necessity of examining the chest whenever cough is complained of, especially in young adults. It is useless to attempt to treat a cough unless we know the cause of the symptom. It is easy enough in many cases to stop a cough by sedatives, but it is by no means always wise to do so. Purely symptomatic treatment is a confession of ignorance or a makeshift of the man who is too hurried to examine. We may get some clue to the cause of the cough from the patient's description of the symptom, and of the character of the expectoration.

In a young person who brings up thick and yellow expectoration with the cough, never omit to

examine the upper part of the chest. A complaint of the usual symptoms of anæmia by a young person whose pale face and lips confirm the history, might lead us to prescribe without examination; but anæmia does not exclude the possibility of tuberculosis, it rather predisposes to this disease. The early detection of tubercular mischief in anæmic subjects is of vital importance, for in my experience the disease in such persons tends to advance rapidly.

We have, in the male corridor, another patient to whom I would draw your attention also in connection with the ætiology of phthisis. He is a footman, 19 years of age. For five years he has been in service in the same family, and for this period he has lived, as male servants are so often obliged to do, in the basement of the house. He describes his bedroom as being close and stuffy, and without a window. I have elsewhere* commented upon the danger of inattention to hygienic requirements in servants' sleeping rooms—a fault which exists even in some of the best town houses. I have had several cases of phthisis in men-servants where the disease has been traceable to the predisposing influence of living in unsuitable rooms. In this patient there is slight deposit in both apices, the left side being the more marked. There is every reason to hope that the patient will improve considerably under treatment.

Quiescent Phthisis.

In this second man, who is shortly leaving the hospital, and whose condition has greatly improved since his admission, we see how the chest falls in when a much damaged lung underneath contracts, as the disease becomes inactive. He is 22 years of age, and has had a cough since an attack of pleuritis in March, 1892. On admission his weight was only 7 st. 4 lb., and he had occasional pyrexia reaching 103° F. There was deficient resonance with fine crackling and tubular breathing over the right apex, and on the left side deficient respiratory movement, dulness almost to the base, with crackling and bubbling râles heard all over that lung.

There was thus very extensive mischief with acute softening in the left lung, which had been previously damaged by the pleurisy.

Now, after about six weeks in the hospital, his weight is 7 st. 10 lb.; his temperature normal or subnormal; he has only a slight cough, and scanty expectoration.

The right side gives much the same signs as on admission.

The left side of the chest is, as you see, much

fallen in; there is cavernous breathing above the scapula and at the base, with whispering pectoriloquy. A slight crackling râle at the apex is the only adventitious sound to be heard. The apex beat of the heart is now 1½ inches outside the nipple line in the fifth space, and there is visible pulsation all over the cardiac area.

On measurement of the chest as high as we can get the tape in the axilla, we see there is a full inch difference between the two sides (right 15½ in.; left 14½ in.). Here the softened tubercle has been got rid of, leaving cavities in the lung. These are diminishing in size by contraction of their walls, and the heart has become uncovered by the retracted lung, whilst the chest wall sinks in. The sequence of events in this case must be regarded as favourable. The mischief gradually dries up, and the signs of softening disappear; cavities are formed, which gradually contract. There is now little or no active mischief. Unfortunately, we cannot call the disease cured; it is only quiescent. It may at any time light up again and extend. But extension would then be less rapid than at first, for when the activity of the tubercular process has been reduced even if its advance be not absolutely checked, the lessened activity leads to fibroid changes instead of the destructive change (softening) of acute tuberculization, and the fibroid tissue acts as a barrier to rapid extension of the disease.

There is much of interest to be noted in the shape of the chest in lung diseases.

Compare these two cases.

In the one (tubercular phthisis) we see a flattened chest, thinly covered. The superior margin of the trapezius muscle stands up as a sharp edge behind the supra-clavicular fossa. The movements of respiration are slight, and chiefly carried out by the lower part of the thoracic walls, and by the diaphragm; the lower part of the lungs have to do extra work.

The other case presents a rounded thorax, also thinly covered with wasted muscles, but the upper part of the body looks heavy, instead of slight and wasted as in the first case. The margin of the trapezius seems to be curved forwards, as though overhanging the supra-clavicular hollow; the shoulders are rounded behind. Here also the movements of respiration are slight, but the whole thorax seems to share equally in the diminished movement. This is a case of fibroid phthisis, the expansion of the lung is deficient throughout, not only at the apex. The auxiliary muscles of respiration acting on the upper part of the thoracic wall are constantly at work, and having developed by use, give the heavy appearance to the upper part of the body. There is a man in the ward with spasmodic asthma, and in him you will see still more marked the heavy shoulders and rounded back, which result from constant effort to draw air into the lungs.

* "The Hygienic Prevention of Consumption." Chas. Griffin & Co., London.

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THE THERAPEUTICS OF THE THYROID GLAND.

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THE therapeutic value of the thyroid gland in the treatment of myxœdema and cretinism may now be considered as completely established. It is not surprising that such a potent remedy in one form of disease should be having an extensive trial at the present time in various other maladies. The results of the latter are still uncertain, although not unhopeful. As is the case with every new remedy, experience has shown us that certain precautions must be adopted to make its administration safe. It is with the view of making the experience already obtained useful to the practitioner, that I have undertaken to write the following short account of the therapeutics of the thyroid gland.

The present successful mode of treatment of myxœdema and cretinism has been reached by three distinct stages.

1. Attempts at transplantation by placing under antiseptic precautions a portion of fresh thyroid gland underneath the skin.

2. Injecting underneath the skin a sterilized liquid extract of the gland.

3. Administering by the mouth, (a) the gland itself, (b) preparations of the gland.

The expectations that the thyroid gland would permit of transplantation were not realized. The absorption of the gland, however, temporarily benefited the patient, and this benefit suggested to Dr. George Murray the second mode of treatment, which proved to be completely successful. It was a method, however, for the success of which two conditions had to be fulfilled:

- (1) That the glandular extract used should be absolutely sterile, and as goes without saying, that the physician should use a perfectly aseptic hypodermic syringe.

- (2) That certain precautions as to dosage and mode of administration were adopted, as alarming symptoms sometimes supervened on the injections if given in the ordinary way.

The third method, that of introducing the remedy into the stomach, for which I am to some extent responsible, has proved so completely satisfactory, and has such obvious advantages over the hypodermic method, that the interest of the latter is now mainly historical.

The introduction of the stomach mode of administration has not only immensely increased the safety and comfort of the patient; but it has saved the medical profession considerable trouble, and has avoided a great deal of unnecessary expenditure of time and money. The manufacture of an extract which could be safely administered hypodermically, must necessarily have involved a considerable outlay of money, and the profession must have remained dependent on one or two firms for its regular supply.

As a rule drugs are administered hypodermically where rapidity of action is of importance. Hence the value of the method for administering Morphia or Ether. Hypodermic injections, I consider, should never be employed in cases where a drug can be administered by the mouth, except where some special advantage is to be gained.

But in myxœdema or cretinism there is no such special advantage. We do not desire rapidity of action. We wish, if anything, to check too rapid change. Rapidity of action is the danger from which we have to steer clear. *Curare cito tute et jucunde* is very well as a general principle, but in practice it sometimes proves an incompatible combination. In myxœdema, *festina lente* should be the rule on account of safety. If it is a question of *curare jucunde* there is no hesitation in saying that a series of hypodermic injections are better avoided.

In the remainder of this article I shall accordingly limit myself to the discussion of the administration of the remedy by the mouth.

In the first place, what is the best form in which to give the drug, if we may call it so? The practitioner has his choice either to deal with the butcher or with the chemist. Some tell me they prefer to get the fresh gland from the butcher, but

the great majority prefer throughout to use the more convenient and elegant preparations of the chemist. There is always an initial difficulty in getting the butcher to supply the correct article, and in getting the thyroid separated for the patient from the surrounding parts. I have been asked by medical men to show them the thyroid gland when the larynx and trachea have already been stripped by the butcher of all their surroundings. I have been shown the thymus, and asked if that was the thyroid. But if the practitioner will only re-acquaint himself with the anatomical position of the gland, and recollect that in the sheep it occupies about the same position as in the human being, that it is a little smaller, and that there is practically no isthmus, he will have no real difficulty in finding it and in ensuring that the patient actually gets it.

To maintain a cure in myxœdema and cretinism or where large doses are desired, as in psoriasis, there is considerable advantage in using the fresh gland, if only on the ground of economy.

If the unsophisticated gland, as it has been called, is to be administered, it should be obtained from the butcher the same day the animal is killed. It may be given uncooked, minced up with jelly or added to beef tea. As it is found that partial cooking does not destroy its activity, it may be given lightly fried. I found a very convenient way to give it was to prepare it as follows: Let it be finely minced and allowed to stand in sufficient cold water to cover it for about half-an-hour. Express through muslin, and add the liquid extract thus prepared to some beef tea.

I may say here that it is very seldom that the sheep's thyroid is found to be diseased. Not more than 2 per cent. have to be rejected by the manufacturers on this account. I should, however, strongly recommend the practitioner to commence the treatment of a case of myxœdema or cretinism with one of the dry or liquid preparations of the gland, for the reason that the dose is so much more perfectly under control, and it is of great importance, as I shall point out later on, to commence with a small dose. The practitioner may be not a little embarrassed to select one from among the many preparations which are now in the market. There are liquid extracts, dry extracts and preparations of the desiccated gland. The curious thing is that they all, as far as I have been able to ascertain, although prepared in very different ways, have a satisfactory action.

The dry preparations are on the whole preferable to the liquid, because they keep better and can be put up in the form of powders, pills, or tabloids, of whatever dose is desired.

I shall briefly mention the various preparations of which I have personal knowledge. One manufacturer prepares an extract in the form of a dry powder on the method devised by Mr. White of St Thomas's Hospital. This has proved very efficacious in a large number of cases under my personal observation. One firm make tablets of thyroid extract, one tablet being the equivalent of five grains of the gland. They also prepare a liquid preparation called Elixir Thyroidin, one fluid ounce of which represents one gland. This is said to keep well without change. The tablets have to my knowledge proved to have an excellent curative effect.

A firm who were the first and practically the only preparers on a large scale of a fluid extract for hypodermic use, supply the same fluid without Carbolic Acid for internal administration, and also manufacture a dry powder from the expressed juice. Their fluid extract is such that 90 minims are the equivalent of the two lobes of a sheep's thyroid gland, while 30 grains of their powder has about the same equivalent. The fluid extract is thoroughly reliable when recently prepared.

Another firm prepare tabloids from the whole gland dried at a low temperature, each tabloid being the equivalent of five grains. I have made considerable use of these and find they also have a perfectly satisfactory therapeutic effect. They can be swallowed like pills, or they can be added to soup, in which they disintegrate completely. Of other manufactures I have no personal knowledge.

I think it matters little which preparation is used, but it should be borne in mind that they probably vary a good deal in potency, and accordingly, general rules for dosage are rather difficult to lay down. As at present there is no official strength or mode of preparation, the most satisfactory method is to state the weight of the gland of which so much of the preparation is the equivalent. As we cannot expect the extract, to be more powerful than the gland itself, we can, with perfect safety, employ it as the equivalent of the corresponding part of the gland.

Individual glands vary, not only in size, but doubtless also in the amount of the active

principle they contain. When extracts are made on a large scale differences of this kind will be adjusted by averaging, and it is quite sufficient to know how many grains or minims of the extract are derived from, say five grains of gland.

I now come to the question of dosage. The first important matter to bear in mind is that the dose for a patient with myxœdema or cretinism must be very much smaller than when the drug is given for other diseases.

The myxœdema patient shows a particular sensitiveness to the drug, so that the quarter of a dose, which would be absolutely without effect upon a healthy man, may produce profound constitutional disturbance, especially if frequently repeated.

We must, therefore, begin with small doses and watch their effect.

Cases of myxœdema or cretinism have been recorded or observed where disastrous results have followed from too large a dose.

For safety, I should recommend not to exceed one twentieth of a gland at first. It is perfectly true that very much larger doses have been given. In my first case I gave two whole glands as a dose, and I have known of a case where a patient with myxœdema has taken, by mistake, as much as six or eight glands at a dose without any serious consequences. But from what we have learnt of the powerful, even lethal, effects of large doses in some cases, it must now be considered unjustifiable to permit any but small doses at first. We must go slowly, feeling our way. The more gradual the change produced the more comfortable will it be for the patient.

The average weight of the two lobes of the sheep's thyroid gland is about eighty or ninety grains. We should, therefore, begin with the equivalent of five grains or so, and give this once a day.

At the end of a week, if no unfavourable symptoms have developed, we can give the same dose morning and evening. In the third week, under the same conditions, we may give it three times a day. It is not likely it will be necessary to give a larger amount than this, and possibly a smaller dose may suffice.

After recovery is established the remedy may be diminished, in frequency or in dose, or in both. There is not now the same danger to be feared from an overdose, but it is well not to employ more of the remedy than is physiologically necessary.

I have given a single dose once a week, and found that to keep the patient in perfect health.

On theoretical grounds one would have expected that a small dose frequently repeated would be preferable to a larger dose less often; but experience shows that a bi-weekly, weekly, or even fortnightly dose is sufficient when once a cure has been made, in order to maintain a proper condition of health. What the proper dose should be at this time will vary with different patients. I found the equivalent of a gland a week to suit very well; but others have found that considerably less was sufficient. It is hardly necessary to say that if the administration of the remedy is suspended, the patient will relapse.

In the *Edinburgh Medical Journal* for February 1894, the further history of a very remarkable case of sporadic cretinism treated by thyroid feeding, by Dr. John Thomson, is given.

The patient was 18 years and 8 months at the beginning of thyroid treatment. His height was then 33½ inches. Twelve months after treatment he had grown 4½ inches, and no one could recognize in the intelligent-looking boy the deplorable object of the past.

Dr. Thomson says that with the exception of the first two or three weeks, when too large a dose was given, it had not been necessary to confine the patient to bed or even to the house. The dose found necessary to maintain the full amount of improvement, was half a thyroid twice a week, the same dose that a year previously made him very ill.

The symptoms which show that an overdose is being given are, rapid action of the heart, rise of temperature above normal, nausea, vomiting, fœtor of the breath, diarrhœa, and general rheumatic pains. If any or all of these symptoms make their appearance the remedy should be suspended for a time, and then given in smaller or less frequent doses. During the improvement which follows the use of the remedy the patient must be treated more or less as an ordinary convalescent, and must be warned against unaccustomed exertion. One or two cases of death from syncope during convalescence were recorded in the early days of the treatment.

The following are the most striking changes which occur under the action of the remedy:

1. A general diminution in the bulk of the body accompanied, as a rule, by a considerable loss of weight.

2. The disappearance of the swelling of the face, hands, and feet, so that the normal features are once more restored.

3. Rise of temperature to normal.

4. Growth of the hair.

5. Recovery of the functions of the skin.

6. Recovery of brain power to a normal state of activity, memory, etc.

7. Recovery of normal menstrual functions.

8. The amount of urine is increased, and the nitrogen contained in it has been found, in one case, to exceed the amount taken as food by about five drachms daily. The increased amount of nitrogen is almost entirely due to increase of urea.

Desquamation of the hands and feet, sometimes as complete as that which occurs after scarlet fever, is a usual event during the process of recovery.

As a rule, the improvement in appearance and in the mental condition are very soon noticeable.

Very old standing cases, and patients advanced in years, are less amenable to treatment than others.

In cretins the same rules should be observed as in myxœdema, except that the doses should be smaller.

The rapid and remarkable loss of bulk and weight which was effected in myxœdematous patients, suggested that a similar beneficial result might be produced by the remedy in the obese. I made an early trial of this in the case of several very corpulent patients, but found no real loss of weight in any of them while taking full doses of the remedy. Dr. Putnam and Dr. Barron, however, have obtained distinct improvement in some cases.

I had high hopes at one time that it would promote the growth of the hair in ordinary baldness. Quite eighteen months ago I tried it in cases of premature baldness, and universal alopecia; I much regretted, however, to find no beneficial result. I tried it in cases of excessive dryness and scalliness of the skin, but did not succeed in producing any effect with even considerable doses.

The administration of rather large doses in these and other cases, without any appreciable physiological action, shows the difference of its effect in health and disease.

Dr. Bramwell has, however, found what, indeed, was to be expected, that idiosyncrasy was exhibited in some cases, and that even a healthy person might be profoundly affected by a very small dose, an observation which shows the need of caution in the beginning of the treatment for any condition.

Dr. Bramwell's successful results in the treatment of psoriasis encourage the hope that the thyroid gland may play an important rôle in the treatment of this and other forms of skin disease. Dr. Bramwell believes that the best method in such cases is to give as large doses as each individual patient can bear. He finds that many such patients can take a quarter of a gland, *i.e.*, about 20 grs. daily for long periods of time. The pulse rate he considers the best indication of overdosing.

It is only right to add that many who have tried the thyroid treatment in psoriasis, have had only isolated cases in which it has appeared to benefit, together with many failures.

In August, 1892, I treated two cases of exophthalmic goitre by thyroid feeding, using large doses, but did not find any very marked result. The idea with which I gave it was to give the patient's thyroid a physiological rest. I referred to these cases in a lecture published in "The Lancet," in 1893. Since then, others have tried the same treatment. The only case where the result is at all convincing, is one published in "The British Medical Journal," in December, 1893, by Mr. D. Owen. On account of the enormous doses given it is so remarkable that it seems almost incredible. The patient, a man of 46, is said to have had by mistake, $\frac{1}{2}$ lb. of thyroids in two days, or about forty whole glands. No very serious results ensued, only dyspeptic symptoms, vertigo, insomnia, and swelling of the face and legs, which soon subsided.

He is said, however, to have been practically cured of his exophthalmic goitre which he had had for twenty years.

The enormous dose taken by mistake is an experiment which one would scarcely like to repeat.

In conclusion, I may summarize the treatment of myxœdema and cretinism as follows:—

(1) Commence tentatively with quite small doses, not exceeding 5 grs. in an adult, or $2\frac{1}{2}$ grs. in a child, given once a day.

(2) Guided by the pulse rate and by the absence of any of the other symptoms, febrile temperature, nausea, vomiting, foetid breath, diarrhoea, rheumatic pains, I have mentioned, which indicate an overdose, gradually increase the amount until about 15 grs. a day are given.

(3) Treat the patient as one would in case of recovery from acute illness, avoiding fatigue or unnecessary strain on the heart.

(4) During convalescence tonics and Cod Liver Oil may be usefully administered.

(5) When the health is re-established, the remedy may be given at longer intervals and in larger doses, from a quarter to a whole gland, or from 20 to 80 grs. a week, in single or divided doses.

In the treatment of maladies of the skin or other conditions,

(1) Begin with a small dose in case of any abnormal susceptibility.

(2) Push the remedy as far as possible without producing the abnormal symptoms already referred to.

A CLINICAL LECTURE

ON

A CASE OF ECLAMPSIA.

Delivered at the Royal Maternity and Simpson Memorial Hospital, Edinburgh, on Feb. 17, 1894,

By DR. BERRY HART.

Physician to the Hospital.

THE patient whose case I wish to discuss with you this forenoon, was admitted to hospital on the evening of the 13th inst.

The following is a brief summary of the history and progress of the case condensed from full notes, drawn up by Dr. D. C. Edington, one of the resident physicians.

Mrs. Maggie Rice, æt. 19, married two years: has had no abortions, and never had fits, scarlet fever, rheumatism, nor any kidney disease. There is no history of oedema, suppression of urine, nor headaches. Confinement expected about 20th February, 1894. Patient caught a chill a week before labour; this gave her a marked cold in the head, and she got rather swollen in the face, but not in trunk nor limbs.

On 13th February, 1894, while at ordinary housework, and feeling very well, she felt at 2 p.m., in small of back and around loins, pain, which a neighbour said was labour. This passed off in a few minutes, but at 2.30 another pain came on, and just then she dropped insensible in a fit, epileptiform in nature, without an aura; the clonic movements, frothing, etc., were marked.

The fit passed off in a few minutes, but she remained unconscious until another came on at 3 p.m. These fits recurred every half hour till 8.10 p.m., all precisely of the same character, but getting worse. At 8.20 p.m., she took the ninth fit since 2.30 p.m., and was admitted here at 8.15 p.m., insensible.

State on admission.—Patient is a strong, heavily-built, and well-nourished woman: face full and congested; no oedema of limbs nor trunk; tongue very foul, dry and tooth-marked: breathing very deep and stridulous: pupils widely dilated, equal.

Temperature 101.4; *Pulse* 136, small, very hard, regular with high tension; *Respirations* 20, full and regular.

Urine, drawn off by catheter, four oz. in amount, brown coloured; sp. gr. 1028, no blood; albumin, 5.8 grs. per oz.; on microscopic examination numerous hyaline and granular casts were found: no epithelial cells.

Condition of uterus.—The os externum admitted little finger; cervix short, thick, and very firm; presentation vertex; no foetal heart; no distinct pains; membranes lax and intact.

Treatment.—She was given ℥j croton oil by mouth, ½ gr. of pilocarpin hypodermically, and 15 grs. chloral. per rectum. The pilocarpin gave very feeble results, but owing to bronchitic condition, it was not at once repeated. In half-an-hour a glycerine and soap enema was given, and just after this the tenth and worst fit of all occurred. Three drops of croton oil was now given in glycerine and water. At 9.40 p.m. Dr. Berry Hart saw her, and ½ gr. morphine hypodermically was ordered. At 10.15 dilatation of cervix digitally was begun, and at 10.45 patient took the 11th and last fit. It was very short, and she then went to sleep. She was now chloroformed, and Dr. Hart bled her to 16 oz. from the arm at 11 p.m. This gave an evident and markedly good result, and she settled to sleep; breathing easy, no stridor, pulse 130, but now full and softer. At 11.40 vaginal douche 1—40 carbolic lotion was given, and then she got another ½ gr. pilocarpin at same time. A large Barnes' bag was put into vagina. Profuse heavy salivation and diaphoresis came on at 12; sleep became very deep, and pupils which had been pinpoint at 11 p.m. were nearly normal now. At 2 a.m. she awoke very restless and struggling, no fit came on, however; at 3.45 she became very wild, almost overpowering attendants: ½ gr. morphine hypodermically put her into deep sleep. At 7 a.m. vaginal bag

removed: cervix almost as before, membranes tense, pains distinct now; *temperature down to 100°; pulse 124, and good.* Cervical dilatation and vaginal douching now resumed till 11 a.m., when bowels began acting copiously.

The membranes were artificially ruptured at 12.30 p.m.; liquor amnii evidently stained with meconium, and gave reactions of bile.

At 5 p.m. on the 14th Dr. Hart delivered her with axis traction forceps, the head being high, and cervix only half dilated, and very tough. Traction on head soon opened cervix, and head passed over perineum at 5.40, occiput behind. Right episiotomy was performed. The child, a male, was dead, with skin peeling. Continuous suture put in the episiotomy wound, but not allowed to involve the skin.

Puerperium.—The patient made a good recovery, but was somewhat dazed for the first three days, with complete loss of memory. The eyes were examined by Dr. George Mackay, but no abnormal condition found. The urine contained mere traces of albumin, and was, soon after labour, secreted copiously.

The child was dead when the patient arrived in hospital, and was born with skin peeling. In some cases the death of the child has been noted as causing the albumin in the urine to disappear. There were no placental infarcts.

This, then, Gentlemen, is the case in brief, and I have now to discuss its nature and give the reasons for the treatment adopted.

The case was one of the ordinary eclamptic nature you will see fairly often in practice. The kidney lesion was probably the parenchymatous nephritis so frequently found in such, and cleared up soon after delivery.

As to the real *nature* of such a case, we are entirely in the dark. It is undoubtedly a great fact that in almost all eclampsia cases we have nephritis, but this is not, in my opinion, the primary cause of the seizures. Undoubtedly, the kidney condition and the fits are usually found coincident in practice, but you occasionally have cases where the urine is unaltered at first

Post-mortem examination of eclampsia cases has demonstrated marked changes in the kidneys, but also hæmorrhages in the liver, as well as in the heart and pancreas. It is remarkable, too, that liver cells, as well as those of the villi, have been found in the circulation.

It is therefore most probable that the essential

feature of eclampsia cases is some blood condition not bacterial—probably chemical and irritant—and it is from its action on the brain, kidney, and liver we get the varied features of such cases. What the change in the blood is we do not know; and neither chemists nor bacteriologists have helped us as yet. Our position in the pathology of eclampsia is this, that beyond the fact of apparently secondary changes in the kidneys, we are completely in the dark as to the essence of the disease.

The blood serum of eclamptic patients has been found more toxic to rabbits than healthy serum, but what makes the difference is unknown. Note that this is a serious statement. Eclampsia occurs once in a hundred and sixty cases, has a mortality of about twenty per cent., and yet obstetricians do not know what the real nature of eclampsia is—all the theories advanced, mechanical or uræmic, have failed us. This does not look well for treatment.

[It may be noted here that the serum of the blood drawn from this patient is being examined by Dr. Stockman.]

From the blood irritant—purely hypothetical as yet—we get irritation of the cortex of the brain, and changes in the kidney and liver usually coincident with it. The kidney changes are best known, and markedly aggravate the case.

The fit in eclampsia is the result of the cortex irritation, and resembles an epileptic one exactly, except that there is hardly ever an "aura," and the sides of the body are not simultaneously affected. The fit is not due to the kidney condition alone. A patient may, if not pregnant, have a worse kidney condition without any convulsions. Between the fits the patient usually lies unconscious and is often quietest before the fit comes on. You make your diagnosis, then, from the nature of the fits, and the kidney changes, as shown by the altered urine, almost always albuminous, and with tube casts. You may have a patient who is pregnant, and who takes an hysterical fit. You will find, however, the urine healthy, and you can easily see that the fit is a sham and has not an epileptiform nature, to say nothing of its want of definiteness. Tetanus you may practically leave out of account.

Remember, however, that the clinical features in such cases are very varied, and that at present I am only discussing an ordinary case of "eclampsia."

In TREATMENT we have to consider three lines

—sedative, eliminant, and the actual treatment of the pregnancy or labour.

Sedative.—We here reduce the irritability of the brain by Chloroform, Chloral, or Morphia. The fits are not due to the kidney condition alone. One may have no fits with complete suppression of urine, and violent fits with only a moderate amount of renal inadequacy. The blood poison, whatever it is, acts violently on the cortex and give us the epileptiform fits. I reiterate this as I wish you to shake yourself clear of the idea that the kidney condition is everything in the case. Now, Chloroform is invaluable as a sedative; it is convenient, rapid, and safe. Its only fault is that it is transient. Chloral *per rectum* is much used in this country, but I have always felt that it might not be retained, and was interfered with by the necessary purgation.

I daresay you would be struck with one feature of the treatment, the administration of two large doses of Morphia hypodermically ($\frac{1}{2}$ gr. and $\frac{1}{3}$). We owe this treatment to Professor Veit, of Bonn, and it is much employed in Germany. Should the fits persist after the first dose, one could give another in a few hours, noting the state of the pupil and pulse. One must not give it in comatose conditions, of course. The result in this case was good, so far as I could judge. At one time Opium was used in eclampsia cases in England (v. "Treatise on Diseases of Woman," by Manning, p. 359. London, 1775), but it has been hardly employed at all during this century. The importance of sedatives to arrest the fits cannot be overestimated, and in prognosis, the fewer the fits the greater the chance of recovery.

For *elimination* of the poison we rely on purgatives, sweating, diuretics, and bleeding. We act as one would do in a uræmic case, but whether we remove the primary poison in this way is speculative.

The best purgative is croton oil, one to three drops, in pill placed on the back of the tongue or mixed with glycerine, and painted over the posterior pharyngeal wall.

To sweat the patient, pilocarpin in $\frac{1}{2}$ gr. doses is admirable. Remember that it is depressing, so watch the pulse, and do not give it in cases with lung cedema. You will find the salivation troublesome unless the patient is kept on her side, and the mouth sponged out occasionally.

You may supplement the action of the pilocarpin by a vapour bath, or by packing the patient

with hot bottles (ordinary beer bottles) placed in stockings which have been wrung out of hot water. When ordinary hot bottles are used see that the nurse does not fill the jar with too hot water and omit wrapping it in flannel, as the patient may get troublesome burns and sores if they lie in contact with the skin.

As to how one is to *treat the pregnancy* in a bad eclampsia, opinions differ. I strongly advise you to induce labour; and if labour is in progress to accelerate it judiciously. Some obstetricians divide the cervix, put on forceps and deliver rapidly, incising the perineum when the head descends. I see no reason for such haste unless the case is desperate. You will find, as a rule, that you can dilate the cervix in a very short time, and then with forceps end the case speedily.

In this case the patient made an excellent recovery, and I was impressed with the favourable action of the morphia and the bleeding. The pint of blood removed made no appreciable alteration in the patient's complexion, and I should be inclined to remove more in plethoric cases. If the idea of a blood irritant be correct, the removal of blood in sufficient quantity should be highly beneficial.

Finally, I wish you to keep in mind the idea that there is something behind the kidney condition.

THERAPEUTICAL NOTES AND FORMULÆ.

Tabes Dorsalis v. G.P.I.—Dr. Lemoine, following M. Garnier, has published a very useful table of the points of differential diagnosis and likeness between these two affections on the following lines:

1. Reflexes. Loss of knee-jerk practically constant in tabes. In G.P.I. the reflex is sometimes lost, but more frequently normal or exaggerated.

2. Sensory disturbances. Lightning pains, very frequent in tabes, only occasionally appear in G.P.I., and then are less violent and more obscure. Visceral crises common in tabes, rare and occasional only in G.P.I. Cutaneous hyperæsthesia and anæsthesia may be found in either malady.

3. Vaso motor troubles. Cerebral congestions exhibited as fits are common in G.P.I. Similar congestions are found in tabes marked in the eyes

and face, but very rarely causing anything like a fit.

4. Trophic disturbances (perforating ulcer and spontaneous fractures) common in tabes, rarer in G.P.I., but in the latter are becoming more frequently reported.

5. Disturbances of special senses. Loss of smell is of great diagnostic value in G.P.I., as also loss of hearing and of taste. Affections of the eyes are more frequent in tabes (Argyll Robertson phenomenon, contracted pupils, ptosis, optic atrophy), with the exception of inequality of the pupils, which is much more frequent in G.P.I.

6. Urinary affections. The incontinence and retention of urine sometimes found in early tabes, are rare in G.P.I., and only develop very late and slowly.

7. Affections of generative capacity. In both diseases impotence is the rule in the later stages, usually preceded by a period of greatly increased sexual power and desire.

8. Motor troubles. The ataxic gait of the tabetic is also found in G.P.I., but in the latter it more closely resembles the gait of drunkenness, and is not so much affected by closure of the eyes. Difficulty in executing small and delicate movements is a marked feature of both diseases. The muscular power retained by the patient in the early stages of either disease, is contrasted with the great loss of power in the later stages of G.P.I. The handwriting of both is very similar.

9. Intellectual disorders. These form the most characteristic distinction between the two diseases. They are early and almost constant in G.P.I. In tabes they are very exceptional indeed, and only appear in the latest stages.

(*Gas. Med. de Paris.*)

Ergot in Neuralgia and Migraine.—Dr. W. H. Thomson, of New York, writes as follows:—"Having for many years relied on ergot in the treatment of migraine, I was led to try it also in cases of neuralgias which were marked by the feature of periodicity. He then quotes full notes of four cases treated with ergot with most successful and gratifying results, a complete cure being obtained in all, after quinine, arsenic, morphia, chloral, etc., had been tried in vain. The most severe case was one that arose after influenza. The method of administration is as follows:—One drachm of the liquid extract is to be taken in water as soon as any pain is noticed,

and the patient is advised to lie down and keep quiet. Three of these doses may be taken in three successive hours, in cases of migraine, if necessary; but for the more common neuralgias the dose is taken in the usual form of three times a day.—(*Med. Rec.*)

Chapped Hands and Face.—A most excellent remedy for chapped hands and face, and one that, if properly used, will cure the most painful cases in from twelve to twenty-four hours, is compounded as follows:

R Tr. Benzoin Co. ℥x
Alcohol ʒij
Aqua Rosæ ℥xxx
Glycerine q. s. ad ʒi ʒj

M. Sig.: Apply to chapped surfaces at night, after they have been washed with soap and warm water, and thoroughly dried.

A second application is rarely required. This remedy is equally efficacious in the treatment of fissured, bleeding, and sore lips.

(W. P. SPRATLING, M.D., *Med. Rec.*)

Creolin is an agent which in infective pustular eczema effectually controls the process. In scaly eczema it affords marked relief to the irritability and itching.

As creolin soap it can be employed with advantage in scabies, impetigo contagiosa, intertrigo, as well as in bromidrosis. The lather should be firmly rubbed into the affected parts several times a day.—(*CARBLOM, Med. Rec.*)

Acne.—

R Camphorated Naphthol,
Resorcin,
Salicylic Acid āā gm. 5
Starch,
Vaseline,
Precipitated Sulphur .. āā gm. 25

M. Et fiat pasta.—(*BESNIER, Med. Rec.*)

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